CERTIFICATE

This is to certify that this dissertation work on ***VATHA KARSANAM*** has been carried out by **Dr.S.UMERA** during the year 2011-2013 in the Post Graduate Department of Maruthuvam, Government Siddha Medical College, Chennai- 600106 under my guidance and supervision in partial fulfillment of regulation laid by **The Tamilnadu Dr. M.G.R Medical University, Chennai** for the final **M.D(siddha) Branch I- MARUTHUVAM** examination to be held in **April 2013**.

This dissertation is a record of original work done and it has not been previously formed the basis for the award of any degree.

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A STUDY ON

VATHA KARSANAM

the dissertation Submitted by

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INTRODUCTION

INTRODUCTION

Among the antique system of medicines, “SIDDHA” system is the south India’s admired system. The word “SIDDHA” is plagiaristic from the word “SIDDHI” which means an entity to be headed for perfection or heavenly bliss. The SIDDHA system of medicine focused on “ASHTAMAHASIDDHI” that is the eight supernatural powers, which helps to attain the **DIVINITY.** They are ANIMA, MAGHIMA, KARIMA LAHIMA, PRAPTHI, PRAHAMIYAM, ESATHUVAM, VASITHUVAM. Those who achieved the above powers are known as “THE GREAT SIDDHARS” .There are many siddhars lived and establish the system. Perhaps 18 of them are known to be important. Those who were called as “18 SIDDHARS”. These legends wrote their knowledge in palm leaf manuscripts. These manuscripts developed as a constructive system of medicine in south India”. By their supreme astuteness, they wrote scriptures on all aspect of life, from arts to science and truth of life to miracle cure.

Among them AGASTHYA is whispered to be a first siddhar directly thought by GOD.

“¾ý ¨É «ÈÂò ¾É ì¦¸¡Õ §¸Êø ¨Ä
¾ý ¨É «ÈÂ¡Áø ¾¡§É ¦¸Î¸ýÈ¡ý”.

- ¾Õ ã Ä÷ ¾ÕÁó¾Ãõ

Know thyself and that makes you free from all evils but, man not knowing his own self becomes victim of all troubles. By preserving the health one can attain the IDOL. That is the goal of our soul.

“ THIRUMOOLAR” one among the 18 siddhars quoted in his book “THIRUMANTHIRAM” which consist of 3000 poems ,mainly about the importance of health that let a way to conserve the healthy soul. He coded the importance of health and elucidate how to prevent the body from diseases. He sophisticated his knowledge to other with the notion that, “A HEALTHY SOUL CAN ONLY DEVELOPED BY A HEALTHY BODY “

“¯¼õÀ¡÷ «ÆÂøø ¯ÂÃ¡÷ «ÆÅ÷
¾¼¼õÀ¼ ¦Áö»¡É õ §ºÃ×õ Á¡ð¼¡÷
¯¼õ¨À ÅÇ÷ì Ì õ ¯À¡Âõ «Èó§¾.
¯¼õ¨À ÅÇ÷§¾ý ¯Â¢÷ÅÇ÷ò §¾§É”

- ¾¢Õ ã Ä÷ ¾¢ÕÁó¾¢Ãõ

Nowadays life style modifications and less physical works lead to many problems which are called as life style modification disorders. These sedentary life style results in **DIABETES, HYPER TENSION, CARDIAC DISEASES, E.T.C.** The global prevalence of diabetes varies from 6.4% to 10.2%.

There are numerous complications accompanied with diabetes. The major and most common complication is **diabetic poly neuropathy.** In siddha system of medicine it’s correlated with **vatha karshanam.**

The disease **“VATHA KARSHANAM**” (DM NEUROPATHY) tingling & numbness of palms & soles ,Glove and stocking type of anesthesia ,Calf muscle tenderness, Flaccid weakness of lower limbs and finally leads to foot drop . It is implicit as sixth and seventh **avathaigal** (complications) of **MADHU MEGAM (**DIABETES MELLITUS**).**

The incident of DM NEUROPATHY is about 60% -70% of world diabetic population having mild to moderate NEUROPATHY. In world’s population about 26% affected by diabetic neuropathy. The severity and incidence of neuropathy are especially great in blacks (3to 6 fold higher than whites.

The complication of VATHA KARSHANAM (DM NEUROPATHY) are tropical foot ulcer, gangrene, neuropathic deformity, non-traumatic amputation. Most serious complication is , life style modification stress leads to isolation.

I presume that there is a correct time to give a very precise reinforcement for a worldwide disease VATHA KARSHANAM (DM NEUROPATHY) by our own soils medicine. Hence the authour prefers the drug “KARUNGALI VER KUDINEER “ to treat disease. Because the KARUNGALI VER (ACACIA CATECHU) has a capacity to reduces blood sugar levels stimulating the beta cells of langerhans in pancreas and stabilizes the capillaries . And it has VITAMIN –B12 which rejuvenates the nerves. Thus i strongly believe that the drug will be a best elucidation for the worldwide disease.

As per siddhars contemplation the diseases should be completely eradicate by a complete diet & life style modification. So i also included the diet regimen is a part of treating **vatha karshanam.(DM NEUROPATHY)**

“ Á¡ÚÀ¡ ÊøÄ¡¾ ¯ ñ Ê ÁÚòÐ ñ ½ı ý
ë ê À¡ Êø ¨ Ä ÔÂı÷ì Ì ”.

-¾ÕÅ û Ù Å÷.

The food which is suitable for our body should be intaken. The food which pervert the ‘thiridhosa’ should be neglected. This is the preventive methods ever since quoted long ago by one of the great saint THIRUVALLUVAR.

"HE WHO TAKES MEDICINE AND NEGLECTS TO DIET WASTES THE SKILL OF HIS DOCTOR"

-CHINESE PROVERB

AIM & OBJECTIVE

AIM & OBJECTIVE

PRIMARY AIM:

To evaluate the safety and efficacy of KARUNGALI VER KUDINEER.

SECONDARY AIM:

The aim of my study is to evaluate the efficacy of the trial drug KARUNGALI VER KUDINEER in VATHA KARSANAM (DM NEUROPATHY)

OBJECTIEVES:

- To amass the literature of both siddha and modern aspect of the disease VATHA KARSHANAM
- To revise the clinical course of the disease VATHA KARSHANAM with deep scrutiny on etiology, patho physiology , pathology, diagnosis, differential diagnosis, complication and treatment by siddha aspect.
- To depict the clinical diagnostic methods practised by siddhars to distinguish the dearrangements of mukkutram, examine the pori pulangal, udal katugal, neerkuri, nei kuri as per the envagai thervugal.
- To have an idea about the incidence of disease with age, sex, occupation, economic status, habits, familial history, previous history, and climate variations
- Detailed clinical investigation
- To study the efficacy of the drug KARUNGALI VER KUDINEER
- To evaluate the safety of the drug
- To find out the biochemical analysis of the trial drug.

REVIEW OF LITERATURE

SIDDHA ASPECTS

SIDDHA ASPECT

In siddha system of medicine, according to the derangements of thiridhosas, (vatham, pittham, iyyam) diseases are classified into 4448 types. The treatment is not only for the physical body's pathology but also for the spiritual mind

Á¸¡Û õ Ì ¨ÈÂ¡Û õ §¿¡ö¦ºöÔõ á§Ä¡÷
ÅÇ¡ Ó¾Ä¡ ± ñ ½¡Â ã ý Ú
-¾¡ÕÅ û Ù Å÷

VATHAM:

Synonyms:

Vayu, vali, Arasan.

Definition:

Vadham or vali is not mere wind but also causes motion, energy and sensation of every cell in the body. Vayu, one amongst the uyir thathukal and panchapootham(five elements of earth) . Human life is classified in to three phases.

- VAZHI- early phase
- Azhal- middle phase
- Iyam- last phase

Å¡¾Á¡ö À¨¼òÐ À¡ò¾ Å ý É¡Â¡ö ¸¡òÐ
§ºòÁ º¾¨Á Ð¨¼òÐ
- §¾Ã ý ÁÕòÐÅ À¡Ã¾õ

Vatham is responsible for edifice of works and movements of various parts of our body. When the vilation of vali kuttram produces

- Pricking
- Gnawing

- Tingling pain
- Loss of functions of affected areas at later stage

DEFINATION:

It is defined as, whenever the vatha kuttram exceeds or decreased in its level it shows the symptoms likes, pricking and irritating pain, disability to do works, tremors, and e.t.c.

ETIOLOGY:

According to yugi sinthamani,

- Excess intake of bitter, sour, and spicy food
- Old food intake
- Long term constipation,
- Hiccough
- Irregular food habbits

VATHA KARSANAM is classified under the 80 types of vatha noi or vazhi noi by the great siddhar YUGI MUNIVAR in his literature YUGI VAITHIYA SINTHAMANI

±ý É§Å Å¡¾ó¾¡ ¦É ñ À¾¡ Ì õ
Á¸ò¾§Ä ÁÉ¾÷¸ Ù ì ¦¸öÐ Á¡Ú
À¢ý É§Å ¦À¡ó¾¨ É§Â §º¡Ã ï ¦ºöÐ
¦À¡¢§Â¡÷¸ û À¢Ã¡Á½ ¨Ãò à „ ½¢òÐõ
Åý É§Å Åî¦º¡ò¾ü §º¡Ã ï ¦ºöÐ
Á¡¾¡À¢¾¡ Ì Õ ¨ Å ÁÈóÐ §À÷ì Ì õ
¸ý É§Å §Å¾ò¨¾ ¿¢ó¨¾¦ºö¾ §À÷ì Ì õ
¸¡Âò¾ü ¸Äó¾ Î §Á Å¡¾ó ¾¡§É.

- À¡¼ø243 Àì¸õ 183

¾¡¦É ýÈ ¸ºô§À¡ Î ÐÅ÷ôÒ ¨ÃôÒ
º¡¾¸Á¡ö Á¢ï Í¸¢Û ï º¨Áò¾ Å ý Éõ
¬¦É ý§È« Å¡È¢óÐ ¦À¡º¢ò¾ Ä¡Öõ
¬¸¡Âò §¾ÈÄÐ Ì Êò¾ Ä¡Öõ
À¡¦É ýÈ À¸ÖÈì¸ Á¢Ã¡Å¢ ÆôÒ
ÀðÊÉ¢§Â Á¢¸×Ú¾ø À¡Ã ¦Áö¾ø
§¾¦É ýÈ ¦Á¡Æ¢Â¡ü§Áü º¢ó¨¾ Â¡¸¢ø
º¡ì¸¢ÃÁ¡ö Å¡¾ÁÐ ¦ºÉ¢ì Ì ó¾¡§É.

- À¡¼ø243 Àì¸õ 183

.....

.....

¬½¡É ÅÃý È¨ É§Â Á¾¢Â¡ Á¡ó¾÷
«¸¾¢ÀÃ §¾º¢Â÷¸ð ¸ý É Á¢Â¡÷
§¸¡É¡É Ì Õ¦Á¡Æ¢¨ Â ÁÈó¾ §À÷¸û
¦¸¡¨Ä¸Ç× ¦À¡ö¸¡Á í Ì È¢ò¾ §À÷ì Ì õ
° É¡É º¼ó¾ý É¢ø Å¡¾õ ÅóÐ
¯üÀÅ¢ì Ì õ §Å¾ò¾¢ Ñ ñ ¨Á ¾¡§É.

- À¡¼ø243 Àì¸õ 183
- ä¸¢ ¨Åò¾¢Â º¢ó¾¡Á½¢(§¿¡ö ¿¡¼ø 600)

According to sadhaga naadi:

- Seasonal attacks (winter season)

¸¼¸Ó¾ø ÐÄ¡õ Å¨ÃÂ¢ø Å¡¾Á¡ Ì õ
¸ñ½¡Ê ¨ÂôÀº¢Ô ÁÐ§Å Â¡ Ì õ
º¾¸ ¿¡Ê(§¿¡ö ¿¡¼ø 596)

According to noai nadal,

- Excess alcohol, toddy intake
- Diabetes mellitus(mega noi)
- Cardiac diseases

ACCORDING TO T.V. SAMBASIVAM PILLAI

Vatha karshanam is a disease which defined as a disease of the foot caused by the vitiated wind humour marked by numbness of the part and inability to bend or stretch and a feeling of something sticking to the foot and enlargement of the limb.

-T.V.SAMBASIVAM PILLAI AGARATHY

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ACCORDING TO THERAYAR VAGADAM:

Å¡Ô §¸¡À¼ò¾¡ø ºóÐ « ¨ ÇóÐ ¾¨ Ä§¿¡Å¡õ
Á¼ì¸Ó÷ ¦¸¡ð¼¡Å¼ Å¼ð¼Ð ¦¸¡¼Â ÁÄí¸ðÎõ
´ì¸¿ÃõÒ ¾¡ý Ó¼í¸ ãÄÁóÐ Å¡ö ¿¼ÕÈ¼ÅÕõ
Á¼ì¸ Ì Ç¼Õõ ¿Îì¸Á¡õ §ÁÉ¼ Ì ý È¼ ÅÕí¸¡§½

-Àì¸õ 76

- Pain in the joints
- Headache
- Excessive yawning
- Constipation
- Excessive salivation
- Burning sensation of the body
- Chillness and tremor.

ACCORDING TO AGASTHIYAR GUNAVAGADAM:

¦¾¡ø¨Ä ¦ºöÂ þý Ûõ¦Å Ì Å¡¾§¿¡ö¸û
¦¾¡øÖÄ¸¼ø Á¡ó¾Õì Ì ¸¡ ñ ÀÐ ñ Î

±ø ¨ ÄÂłøÄ¡ Å¡¾§¿¡ö¸û §¿÷¨ Á¾ý ¨ Á
þÂøÀ¡¸ «Èłó¾¼§Å ÅłÀÃí§¸§Ç

ÅłÅÃÁ¼¡ «°¾ºý Élà ¨Ç §¿¡×
Å¡łÅ¡É ã¨ÇÂÐ ÁłÕÐÅ¡¸ł
«ÅÉł¾Élø ¾¼Á¡¸ô §À¡Å¾¡Öõ
«ôÀ§É ãò¾ÃÌ Ì ñÊì¸¡ö ÅłÂ¡¾Â¡Öõ

¾¡ÅÓÉłÅ÷¾÷¸¡ì¨¸ §Á¸§Ã¡¸õ
¾ý¨ÁÔûÇ Óò¾ ñ Îì¦¸¡Ê ÅłÂ¡¾
«ÅÁłÄ¡ô À¡¡ºø ¿ÃõÀØò¾í¸ ñ¼¡ö
« Ï Ì Á¼¡ Å¡¾§¿¡ö ¬ Ìõ À¡§Ã

« Ï Ì Á¼¡ Á¡Áºò¾ý ÅłÂ¡¾Â¡Öõ
«ôÀ§É Ý¾¸ò¾ý ¦ÀÕì¸¡Öõ
Ì½ÁłøÄ¡ þÃºõ Åí¸õ ¾ý É¡Öõ
ÌÊ¦¸Î ò¾ Å¡¾ÁÐ ¯ ñ¼¡ÁôÀ¡

- Àì¸õ 16

- Brain disease
- Kidney diseases
- Sexually transmitted diseases
- Vertebral column & spinal diseases
- Menorrhagia

ACCORDING TO THERAYAR MAHAKARISAL,

¬¸í¸Úì Ì §¿¡ Â¡¸óÐÊì Ìõ
¬È¡ò¾ ¦Âý É¦Áö §Â¸í ¦¸¡¾ì Ìõ

¬ Õ¦Áö ÅŧÂ÷¾ŧÁŧ÷ Á¾ó¾õ Å¡ö ã î Í
¬ Ì §ÁÂŧÐ Å¡¾ §ÁÄŧÉŧ

- Àì¸õ 15

- Discolouration of normal skin
- Burning sensation of body
- Sweating
- Numbness
- Dyspnoea

ACCORDING TOPARARASASEKARAM:

¦¾¡Æŧø ¦ÀÚ¨¸ôÒì ¸¡÷ò¾ø ÐÅ÷ò¾ø Åŧ ï Í¸ŧ Ï ï §º¡Úõ
À¨ÆÂ¾¡õ ÅÃÌ Áü¨ÈÂô ¨Àó¾ŧ¨ ½ ÂÕó¾ŧÉ¡Öõ
±Æŧø ¦ÀÈô À¸ÖÈí¸ŧ þÃÅŧÉŧ ÖÈí¸¡¾ ¾¡Öõ
Á¨Æ¿ŧ¸÷ ÌÆÄŧÉ¡§Ç Å¡¾í§¸¡ Àŧì Ìõ ¸¡§½.
-Àì¸õ-12

- Consumption of excessive
- Astringent
- Savouries
- Cereals
- Rancid food
- Day time sleep
- Lacking night sleep
- Increases vatha kuttram

ACCORDING TO AGASTHIYAR GUNAVAGADAM:

« õÒÅŧÂŧø Å¡¾§¿¡ö ÅÕ Ì õ §¿÷¨Á
« ôÀ§É ¦º¡øÖ¸ŧ§È ÉÈŧÅ¡öì §¸Ù

¦¾õÒ¼§É ´¡¼ò¾ §ÄÛ ÁôÀ¡
¦¾ÇŢŢå¡¸ô ÀÄÅ¼ò¾ §ÄÛ ÁôÀ¡
ºõÀŢÃÁ¡öô À¡ºº ¿ÃõÀŢ §ÄÛ
º¾ŢÃ¡É ºÄÉ ¿ÃõÀŢ §ÄÛ ÁôÀ¡
¸õÀŢ¾Á¡ ÂŢÃ ñ Î ÁøÄ¡Áü §À¡É¡ø
¸Ê¾¡É Š¾õÀÉ§Ã¡¸ ¦Áý§È ¦º¡øÅ¡ö

- À¡¼ø121 Àì¸õ31

- This poet is the evidence for, that vadha diseases may occur in a single or many places.
- Vatham may affect both the sensory and motor nerves.

ACCORDING TO AGASTHIYAR KANMA KANDAM300:

á¦Äý È Å¡¾õ Åó¾Å¨¸ ¾¡§ÉÐ
Ñ ñ ¨ÁÂ¡ö ì ¸ýÁò¾ý Å¨¸¨Â§¸Ù
¸¡ÄŢ§Ä §¾¡ý È¢ÂÐ ¸Î ôÀ §¾Ð
¨¸¸¡Ä¢§Ä Ó¼í¸¢ÂÐ Å¢ì¸¦ÁÐ
§¸¡Ä¢§Ä À Î ì¸¢ý È Å¢ÕðºÁ¡É
Ì Æó¨¾ ÁÃó¾¨ É ¦Åð¼ø §Áø§¾¡ø º¢Åø
¿¡Ç¢§Ä º¢Å¦ºóÐ ¸¡ø ÓÈ¢ò¾ø
¿øÄ ¦¸¡õÒ ¾¨ÆÓÈ¢ò¾ø ¿øò¾ø ¾¡§É

- Àì¸õ13

- Removing the barks of living trees
- Causes grievous injuries to animals

are the reasons for causing vatha diseases.

ACCORDING TO KANNUSAMYAM:

§Á¸ò¾ø ¿¡¦Æ¦× §Á×Á¾ø Å¡¾§¿¡ö.....

-À¡¼ø 59, Àì¸õ18

In megha rogam, madhumegam(diabetes mellitus) causes vatha noi.

ACCORDING TO THERAYAR SEKARAPPA:

À¦½¦Âð¼Å¨½ ¦ºôÒ§Åý ¸Õô¦À¡ÕÇ¦
ÁÐ§Á¸¦Á¡¦§Ã¡¸í Å¡¾.....

- Àì¸õ45

NOI EN (CLASSIFICSATIONS):

The classification of vatha diseases is varied amongst the siddhars. Some them re quoted below

- According to yugivaithiya sinthamani, vatha diseases are classified in to 80 types .

“ ±ý É§Å Å¡¾ÁÐ ± ñ À¾¡ Ì õ
²üÈÁ¡õ §ÀÕ¨¼Â ±Æ¦¨Äì §¸Ç¡ö
Åý É§Å Å¡¾Š¾õÀõ Å¡¾¸÷„½õ
Å¨¸Â¡É ¸ÃŠ¾õÀó ¾ÄŠ¾õ Àó¾¡ý
¦À¡ý É§Å âÍÅ¡ ¾ò¾¦§É¡Î
¦À¡¦Â¸¡ Ç¡ï º¸Ó ãÕ Š¾õÀõ
Ì ý É§Å Å¡¾¸÷ý Éí Ì¡¦„ ¸¦¡¦„õ
ÜÈ¡É ¿¡¦ò¾¨ÄÂ¦ý Å¡¾Á¡§Á”

¾¨Äì ÌõÀ Å¡¾¦Á¡Î Á¨Äò¾ ¸õÀõ
¾Õì¸¡É À¡½¦¸õÀí ÜÉ¦Å¡¾õ
«¨ÄÂ¬ Ì É¦Å¡¾õ «§„À¸ó¾¡ý

« ¾Å¡¾õ ¯À¸¾Óõ ¦¿üÈ Ý ¨ Ä
¸¨ ÄÂ¡É Ý ¨ Ä¦ºÅ À Î Å¡ Â¡ Ì õ
¸ Î À ì ¸ Å¡¾¦Á¡ Î À ‡ Å¡¾õ
¾ ¨ ÄÂ§Àö Å¡¾¦Á¡ Î À Ã¡½¡ ÄÂó¾¡ ý
¾Õ ì ¸¡É ºÄŠ¾õÀ¦Á¡ Î ºóÐÅ¡¾ Á¡§Á.

ºóÐÅ¡ ¾ò§¾¡ Î º¸É Å¡¾õ
¾¡ò¾Åü Ò¾Å¡¾ ÓÃ¸ ý Å¡¾õ
¯óÐÓÃ ¸¡§Â¡ Î Å¡ŠÅ ¾õÀõ
¯Ú¾À¡ ºò¾õÀõ §¿ò¾Ã À×ò¾Ãõ
« óÐ¾ ñ ¼ Å¡¾¦Á¡ Î Å‰¼ò ¾ó¾Ãõ
« ¾ºÉ¡ Å¡¾¦Á¡ Î Á¸¡Å¡ ¾ó¾¡ ý
ÓóÐÁÕóÐ Å¡¾§Á¡ Î ¾Ã Å¡¾õ
Ó¸ú ã ò ¾Ã×¾Ã Å¡¾Á¡ ¦Á ý § É.

± ý È Í ì ¸Ä Å¡¾ ï ºù ÅÃí ¸ Å¡¾õ
±ÆÖ ° ÷òÐÅ Å¡¾¦Á¡ Î Å ï » ¡É ò ¾õÀó
¦¾ ý Èº Ãì ¸õÀÅ¡¾ Ó ï º¾Š ¾õÀõ
¦ºÂÅî º Ã å À¦Á¡ Î ¸ ñ ¼ì ¸Ã¡¸õ
¿ ý È¾¡õ ¿¸¡¡¦Â¡ Î À¾¾ Å¡¾õ
¿Ä§Â¡É Ý ¨ Ä¦Â¡ Î ¦¸÷ôÀ Ý ¨ Ä
Ì ý ÈÅ‰¼ Ý ¨ Ä¦Â¡ Î Ì ¼øÅ¡ ¾ó¾¡ ý
Ì È Â Í Å Å¡¾¦Á î Í Å¡¾ó ¾¡§É.

¾¡ ý ã Î Å¡¾Á¡õ Å Í Å¡ ¾ó¾¡ ý
¾ Û À ƒ ò ¾õÀ¦Á¡ Î ¾ó¾Ã ¦ÅðÊ
Å¡ ý Å¡¾ Í §Ã¡ ½¾ ï ºòÐÅ¡¾ Í §Ã¡ ½¾õ
Á¸ò¾¡É ¨ Å¸¾ Å¡¾î Í §Ã¡ ½¾õ
° Û ¾Ã Å¡¾ Í §Ã¡ ½¾ó¾ý §É¡ Î
¯ÚÀÂò¾Â Å¡¾ Í §Ã¡ ½¾Ó Á¡ Ì õ
§¾ ý º§Äð Î Á Å¡¾ Í §Ã¡ ½¾ó¾¡ ý Á ì ¸Š
ºóòÐ¾Ã Å¡¾ Í §Ã¡ ½¾Ó í ¸¡§½.

¸¡ ½ §Å Ì ½ ÅÅ ¾¡É¸Á¡õ Å¡¾ í
¸ ñ ¼¸Å ¾¡É¸Á¡õ Å¡¾ Á¡ Ì ó

§¾½§Å º¢Ãì¸õÀ ÅÅ¾¡É ¸ó¾¡ý
º¡È¢Â§¾¡÷ Å¡¾ò¾¢ý È¨Ä§¿¡ì¸¡Î
â½§Å À¢ò¾ò¾¢ý È¨Ä§¿¡ì¸¡Î
Ò¸Æ¡É º¢§Äð ÎÁò¾¢ý È¨Ä§¿¡ì¸¡Î
º¡½§Å ºý É¢Å¡¾ò¾¢ý È¨Ä§¿¡ì¸¡Î
¾ÕÃò¾ À¢ò¾ò¾¢ý È¨Ä§¿¡ì¸¡§¼.

§¿¡ì¸¡É ¸¢ÕÁ¢¸ó¾ ¾¨Ä§¿¡ì¸¡Î
Ñ¾üÝ¡Â¡ Å÷ò¾¦Á¡ Î ºó¾¢Ã¡ Å÷ò¾õ
° ì¸¡ý ¸÷É¡Å¡ ¾ó¾ý §É¡Î
´Õ¾¨ÄÂ¢ý Å÷ò¾Å¡ ¾Ó§Á Â¡Ìõ
Å¡ì¸¡É Å¡¾¸÷É Ý¨Ä §Â¡Î
ÁÕÅ¢Â§¾¡÷ À¢ò¾¸÷É Ý¨Ä Â¡Ìó
§¾ì¸¡É º¢§Äð Î Á¸÷É Ý¨Ä §Â¡Î
¦ºÂÁ¡É ¸¢ÕÁ¢¸÷É Ý¨Ä ¾¡§É.

¾¡É¡É ¾ó¾Å¡ ÔÅ¢ýÈý §É¡Î
¾¡ì¸¡É Å¡¾ó¾¡ý ± ñ À¾¡Ìõ.

- **Bogar classified into 80 types**

Å¡î¦ºýÈ Å¡¾õ ± ñ ÀÐ×õ §À¡Ìõ

- §À¡¸÷ ¨ Åò¾¢Âõ 700

- **ACCORDING TO PARARASASEGARAM:**

µ¾¢Â Å¡¾ ¦Á ñ ô ¾¢ý Ì½ ÓÚ§À÷

-À¡ÃÃ¡º§º¸Ãõ

- **ACCORING TO AGASTHIYAR:**

± ñ ÀÐ Å¡¾Á¡ Ì ÁìÕÅ ¨ ¸ôÀ Î ò¾ì ¸¡ ½ì ý
¿ý ÒÚ « ¨ Ãì Ì §Á§Ä ¿¡üÀÐ Å¡¾Á¡ Ì õ
À ñ §ºÃ ¨ Ãì Ì ¸¦§Æ ÀòÐ ¿¡ ý ¸¡ Ì õ ¦Áý Ú
Å ñ Î §º÷ Ì ÆÄìÉ ¡§Ç Å¡¾ò¾ì ý Ü Ú¾¡§É

- « ¸ ò¾Â÷ 2000

ÁüÈ§Á Å¡¾§Ã¡ ¸õ Å ¨ ¸ ± ñ ÀòÐ ¿¡§Ä

- « ¸ ò¾Â÷ Ãò¾ì É Í Õì ¸õ

in,agasthiyar guru nadi nool,

vadha diseases are classified as 84 types.

ACCORDING TO YUGI in his another book :

¬ ÁôÀ¡ Å¡¾õ ¦¾ ñ ÀòÐ ¿¡Ö
« ¾Û ¨ ¼Â Ì ½ ¡ Ì ½ í ¸ Ä¼ í ¸Ä¡ ¸

- ä ¸ì ¨ Åò¾Â ºì ó¾¡Á½ì 800

- In JEEVA RATSHAAMIRTHAM - 80 types
- In THERAYAR GUNAVAGADAM-81 types
- In, SARAGASAMHITHAI-2nd part -80 types
- In, ASHTANGA SANGHIRAGAM-85 types.

VATHA KARSANAM

Vatha karsanam is one among the 80 types of vatha noigal. The major symptoms are pain and numbness over the sole of the foot, irritating pain in both extremities that pain radiates to the whole parts of body

Å¡¾ ¸÷º É õ

À¡÷ì ¸¢ý È Å¡¾× û ÇÊÂ¢ü º¡ ½¢
À¾¢òÐ ¨ Åò ¾Ð§À¡Äô À¡¾ ¦Á í Ì õ
§¿¡ ì ¸¢ý È Ì ¾¢ÉÃõÒì ¸¡ø¸ ¦Ç í Ì õ
¦¸¡Ê¾¡É À¡ÃÁ¡öò ¾¢Á÷ô Ò ñ ¼¡¸¢
Å¡÷ì ¸¢ý È Å¡÷ò ¨ ¾¸ û Á¢¸§Å ¦ºöÐ
Å ¨ Çó¾¢Ê Û õ ¿¢Á÷ó¾¢Ê Û õ Åº í §¸¡ ¼¡Áø
²÷ì ¸¢ý È ¸¡Ö ¨ Çì Ì õ Å¡¾ ¸÷º É õ
®¾ÄÈ Á¢øÄ¡¾¡÷ì ¦¸öÐ í ì ¸¡§½.

± ý À¾¡ø, þ¾¢ø ¸¡ÄÊÂ¢ü º¡ ½¢ ¨ Âô â º¢ÂÐ §À¡ ý Ú « ÕÅÕô ¨ À ¯ ñ ¼¡ ì ¸¢ì Ì ¾¢ì ¸¡ø¸ ¿ÃõÀ¢ø ÅÄ¢Ôõ, ¸¡ø¸ û ¸ÉÁ¡ ¸×õ, ¸¡Ä¢ø ÅÄ¢Ôõ ¾Á¢Õõ ¸¡ Ï õ. ¯¼ ¨ Ä Å ¨ Çì ¸×õ ¿¢Á÷ì ¸×õ ÓÊÂ¡¾Å¡Ú §¿¡ Ì õ.

According to tamil lexicon dictionary vol II

KARSANAM means - burning sensation

- ¸¡ÄÊÂ¢ü º¡ ½¢ ¨ Âô â º¢ÂÐ §À¡ ý Ú « ÕÅÕô ¨ À-numbness over the soles
- Ì ¾¢ì ¸¡ø¸ ¿ÃõÀ¢ø ÅÄ¢ - nerve pain in lower limbs.
- À¡ÃÁ¡ö ¾¢Á÷ôÒ ñ ¼ì ¸¢ -numbness over the palms and soles
- ¸¡Ö ¨ Çì Ì õ- burning sensation

MUKKUTRA VAERUPADUGAL :(Pathogenesis)

¯ûÇ§¾¡÷ ¯¼Ä¢ý ÜÚ
¯ÚôÒ¸ Ù¼ý Å¢ÃÅ ¿¢ý Ú
ÓüÚ§Á §¿¡ö¸û Â¡×õ
Ó¾Ä¢É¢§Ä §¾¡ý Úõ §À¡Ð
ÀüÚ§Á Å¡¾ À¢ò¾
º¢§ÄüÀÉó¾Éó ¾ý É¢ø «Åü¨È
ÀüÈ¢§Â §¾¡ý Ú ¦Áý Ú
À¸÷¾É÷ Ó É¢Å÷ ¾¡§Á
-« ì ò¾¢Â÷ Ì Õ ¿¡Ê

Disease occurs due to the derangement in

- Uyir thathukkal
- Udalthathukkal
- kala marupadu(seasonal changes)
- Thinai(living lands) and
- Udal vanmai.

Mukkutra Iyal :

The function of the three uyir thathus:

a) **Vali – (Kattru + Veli)**

b) **Azhal – (Thee)**

c) **Iyyam – (Neer+Mann)**

The alteration of three thathu in their reaction to extrinsic or intrinsic factors results in disharmony. This altered harmony and balance variation of the three thathus results in disease. Their natural ratio (1 :½:¼) to each other is discerned by the

physician at the wrist and each nadi is individually assessed for its strength, speed and regularity.

The following poem describes the origin of three uyir thathus

‘,Ug;ghd ehb vOgNjhBuh

apukhd Njfj;jpy; Vyg; -ngUehb

xf;fj; jrkj; njhopiy a+f;f jrthAf;fs;

jf;fgbahdNj rhu;G”

‘rhUe; jrehb jd;dpy; %yk; %d;W

NgUkplkp gpq;fiyAk; gpd;dYld;- khWk;

ciuf;ftpuw; fhw;nwhl;Lzu;j;J Nkehrp

The three Thathus are manifested at the wrist and are individually and collectively assessed. These three humour are divided in to various types and have their functions specifically.

VATHAM

FUNCTIONS OF VALI:

‘ஒழுங்குடள் தாதேழ்மூச் சோங்கி இயங்க

எழுச்சிபெற எப்பணியும் ஆற்ற - எழுந்கிரிய

வேகம் புலன்களுக்கு மேவச் சுறுசுறுப்பு

வாகளிக்கும் மாந்தர்க்கு வாயு”

- மருத்துவ தனிப்பாடல்

The term vatham denotes vayu, dryness, pain and flatulence. Based on functions and locations it is classified in to 10types. They are tabulated below

S.NO	VATHAM	GENERAL FEATURES	Changes in
1.	Piranan	Responsible for respiration and it is necessary for proper digestion	Normal
2.	Abanan	Responsible for all downward forces such as voiding of urine, stools, semen, menstrual flow	Affected due to poly uria.
3.	Viyanan(paravukaal)	Dwells in the skin and is concerned with the sense of touch... extension and flexion of the parts of the body and distribution, of the nutrients to various parts of the body	Affected due to pain & numbness
4.	Uthanan (melnokkukaal)	Responsible for all kinds of upward motion such as nausea, vomiting etc...	Normal
5.	Samanan(nadukkaal)	Considered essential for proper digestion, assimilation and carries the digested nutrients to each and every organ	Affected due to poly phagia
6.	Nagan	Helps in opening &closing of eyelids	Normal
7.	Koorman	Responsible for vision, lacrimation and yawning	Affected due to vision impairment

8.	Kirugaran	Induces appetite, salivation, all secretions in the body including nasal secretion and sneezing	Normal
9.	Thevathathan	Induces and stimulates a person to become alert, get anger, to quarrel, to sleep etc	Affected due to weakness
10.	Dhananjeyan	Resides in the cranium and produces bloating of the body after death. This leaves from the body after 3days of death, forming a way through the skull.	--------

PITHAM

FUNTIONS OF AZHAL:

‘பசிதாகம் ஓங்கொளிகண் பார்வைபண் டத்து

ருசிதெரி சத்தி வெம்மை வரம் - உசித

மதிகூர்த்த புத்திவனப் பளித்துக் காக்கும்

அதிகாரி யாங்கா னழல்”

மருத்துவ தனிப்பாடல் பக்கம்16

It is the thermal life force of the body. It is subdivided into five types. They are

S.NO	PITHAM	NORMAL FEATURES	CHANGES IN
1.	Anarpitham	Peps up the appetite and aids in digestion.	Affected due to poly phagia
2.	Ranjagapitham	Responsible for the colour and contents of blood.	Affected due to anaemia
3.	Sathagapitham	Controls the whole body and is held responsible for fulfilling a purpose.	Affected due to generalised disability
4.	Pirasagapitham	Dwells in the skin and concerned with the shine, glow, texture and its complexion	Normal
5.	Alosagapitham	Responsible for the perception of vision.	Affected due to vision disturbance

KABHAM

FUNTIONS OF IYAM:

‘திடமீயு மென்பிணைப்புத் திண்மையுற்ற யாப்பும்

அடலேர் வழுவழுப்பும் ஆக்கைக் - கிடர்க்கு

வெருவாப் பொறுமையும் மேலான காப்பாம்

பெருமைத்தா மையமெனப் பேசு”

-மருத்துவ தனிப்பாடல் பக்கம்20

It is responsible for the stream lined functions of the body and maintains body’s defence mechanism intact. It is again classified into 5 types.

S.NO	KABHAM	GENERAL FEATURES	CHANGES IN
1.	Avalambagam	Lies in the respiratory organs, exercises authority over other khapas and controls the heart and circulatory system.	Normal
2.	Kilethagam	Found in stomach as its seat, moistens the food, softens and helps to be digested.	Affected due to poly phagia
3.	Pothagam	Hold responsible for the sensory perception of teste.	Normal
4.	Tharpagam	Presents in the head and is responsible for the coolness of the eyes, sometimes may be referred to as cerebrospinal fluid	Affected due to vision impairment
5.	Santhigam	Necessary for the lubrication and the free movements of joints.	Affected due to joint pain

UDAL KATTUGAL

S.NO	UDAL KATTUGAL	GENERAL FEATURES	CHANGES IN
1.	Saaram (digestive essence)	Responsible for the growth& development. It keeps the individual in good temperament and it enriches the bood.	Deranged due to physical and mental disability
2.	Senneer (blood)	Responsible for the colour of blood and for the intellect, nourishment, strength, vigour and valour of the body.	Deranged due to increased blood sugar
3.	Oon (muscle)	Gives lookable contour to the body as needed for the physical activity. It feed the fat next day and gives a sort of plumpness to the body	deranged
4.	Kozhuppu (fat)	Lubricates the organs to facilitate frictionless functions.	Normal
5.	Enbu (bones)	Supports & protects the vital organs, gives the definite structure of the body and responsible for the posture and movements of the body	Affected in joint pain in some patients
6.	Moolai (bone marrow)	Nourishes the bone marrow and brain which is the centre that controls other systems of body	Normal
7.	Sukkilam/ Suronitham(sperm/ ova)	Responsible for reproduction	Normal

PARUVAKALAM

S.NO	PERUM POZHUTHUGAL	MUKKUTTRA MARUPAADUGAL
1.	Kaar kaalam (Aavani & purattasi) Aug 16 to Oct15	VATHAM-vettunilai vazharchi PITHAM-thanilai vazharchi
2.	Koothir kaalam (Iypasi &karthigai) Oct 16 to Dec15	VATHAM- thanilai vazharchi PITHAM- vettunilai vazharchi
3.	Munpani kaalam (Margazhi & Thai) Dec16 to Feb15	PITHAM- thanilai vazharchi
4.	Pinpani kaalam (Masi& Panguni) Feb16 to June15	KABHAM- thanilai vazharchi
5.	Elavenir kaalam (chithirai & vaikaasi) April16 to June15	KABHAM- vettunilai vazharchi
6.	Mudhuvenir kaalam Aani & Aadi June16 to Aug 15	VATHAM- thanilai vazharchi

THINAI (LAND):

Siddhars classified the lands in to five types. They are

1. Kurunchi - Mountain range
2. Mullai -Pastoral area of the forest
3. Marudham -The fertile river bed
4. Neidhal -The coastal region
5. Paalai - Arid desert

RELATION BETWEEN MUKKUTRAM, KAALANGAL AND THINNAIGAL

VATHAM	Mudhuvenil kalam	Kaar kalam	Koothir kalam	Vatha disease is more prevalent in **Neidhal** land
PITHAM	Kaar kalam	Koothir kalam	Munpani	Pitha disease is more prevalent in **Mullai** land
KAPHAM	Pinpani	Elavenil kalam	Mudhuvenil kalam	Kaphadisease is more prevalent in **Kurunchi** land

UDAL VANMAI (IMMUNITY):

Siddhars classify Udal vanmai as three types. They are

1. Iyarkai vanmai
2. Kala vanmai
3. Seyarkai vanmai

Since VATHA KARSANAM patients are suffering with pain as principal symptom, we came to understand that it is because of alteration in Vali thathu and Vali should be the primary causative factor (Muthanmai kutram). It can be confirmed by the words of great Siddhar Therayer

“Å¡¾ÁÄ¡Đ §ÁÉ¢ ¦¸¡¼Đ”

PINIYARI MURAIMAI (DIAGNOSIS):

It means the method of diagnosing the disease.

‘மதித்திடற்கருமை வாய்ந்த

மாண்பரிகாரமெல்லாந்

துதித்திட வுணர்ந்தானேனுந்

Jfswg; gzpapd;wd;ik

gjpj;jpl Tzuhdhfpw;

gaDwhdhfhyhNd

tpjpj;jpL gpzpj;jpwj;ij

tpsk;GJ Kjw;fz;kd;Ndh”

- rpfpr;rh uj;jpdjPgk;- gf;fk; 3

The above poem describes that diagnosis is very important for the physician to treat the disease.

Four steps are followed in diagnosing the disease. They are,

a. Poriyaal arithal

b. Pulanal therthal

c. Vinaathal

d. Envagaithervu

In detail,

a. Poriyaal arithal:

In this the physician should carefully observe the changes that occur in the five sensory organs (Porigal) of the patient.

b.Pulanal therthal:

The physician carefully applies his five senses of perception, smell, taste, vision, touch and sound to understand the condition of the patient.

c.Vinaathal:

The physician should interrogate about the patients name, age, occupation, socio economic status, food habits, history of past illness, history of present illness, family history, diabetic history and frequency of symptoms.

d.ENVAGAI THERVUKAL

‘eh epwk; nkhop tpop ky%j;jpuk;

ehb guprkpit kUj;JtuhAjk;”

-Neha;ehly; Neha; Kjdhly;-253

ehbahy; Kd;Ndhu;nrhd;d
ey;nyhypguprj;jhYk;

ePba tpopapdhYk; epd;w
ehf;Fwpg;gpdhYk;

thba NkdpahYk; kynkhL ePupdhYk; #ba
tpahjpjd;idr; RfKld;mwpe;JghNu”

- jpUke;jpuk; -10 jpUkiw

Nowadays advanced diagnostic tools have been developed by modern bio-medical scientists. But Siddhars have given eight diagnostic methodological tools. They are called as Envagai thervu.

Eight fold system of clinical assessments

Siddhars have given eight diagnostic methodological tools. They are,

1. Naa
2. Niram
3. Mozhi
4. Vizhi
5. Malam
6. Moothiram
7. Naadi
8. Parisam

GENERAL FINDINGS:

1. **NAA:**
    i. Signs and symptoms in the tongue are noted here.
    ii. Color, salivary secretion, ulcers, coating, inflammation, taste changes, deviation and its nature are generally noted.

In *VATHA KARSANAM* the naa may be affected due to the pallor of the tongue and dryness of mouth due to polyuria.

2. NIRAM:
    i. The color of the skin is noted here.

In *VATHA KARSANAM* the niram may be affected due to the pallor of the body and sometimes hyper pigmentation of body.

3. **MOZHI:**
    ii. Character of the speech is noted, mainly uratha olli(high pitched), thazhntha olli(low pitched), or resembles the sound of any instrument.

In *VATHA KARSANAM* the mozhi will be affected to the patients who have severe pain leading to the thazhntha olli

4. VIZHI:
    iii. Character of the eye is noted. Color, warm, burning sensation, irritation, visual Perception.

In *VATHA KARSANAM* the vizhi may be affected due to the pallor of the lower eyelid, vision disturbances' like phrespiobhia, cataract (bilateral& unilateral).

5. MALAM:

iv. The stools are examined for quantity; hardening (malakattu), loose motion (bethi), Color and smell.

In *VATHA KARSANAM* the malam will be affected due to either constipation or diarrhea.

6. **MOOTHIRAM:**

a. Neerkuri:

v. The urine is examined for its color, odour, volume, froth and weight.

In *VATHA KARSANAM t*he moothiram will be affected due to polyuria, burning micturation, urgency of urine.

b.NEIKURI

‘mUe;J khwp ujKk; mtpNuhjkjha;

mf;fy; myu;jy;mfhyt+d; jtpu;jow;

Fw;wstUe;jp cwq;fp itfiw

Mbf;fyrj; jhtpNa fhJnga;

njhUK$u;j;jf; fiyf;Fl;gL ePupd;

epwf;Fwp nea;Fwp epUkpj;jy; flNd”

-rpj;j kUj;Jthq;fr; RUf;fk; gf;fk;509

The early morning urine of the patient is analyzed by dropping a drop of gingely oil on the surface of the urine sample. The accumulation, formations, changes, and dispersal under the sunlight without any external disturbances of the urine sample can be noted.

- Vatha neer - The oil spreads like snake
- Pitha neer - The oil spreads like ring
- Kapha neer - The oil spreads like pearl
- If the oil spreads gradually, it indicates good prognosis
- If the oil spreads fast or gets mixed completely with urine or sinks in urine, it suggests bad prognosis.

Since *VATHA KARSANAM* is due to the derangement of vatham and pitham,the neikuri will be vatha or pitha neer.

7. NAADI:

Naadi is a Unique Siddha Pulse reading .

À‡ò¾ò¾‡ø Å¡¾Á¡¸‡ø À‡¼¡‡Ô í ¸¡Ö í ¨ ¸Ô í
Ì ò¾Ð §À¡§ÄÂ¡ Ì õ.....
« ò¾‡Â¡ÔÄÕ§Á É‡.....
-« ¸ò¾‡Â ¿¡Ê

º¡üÈÀ‡ò ¾ò¾‡ø ºÐÃ¡¸ Å¡¾ÓÈ
²üÈì ¸¡Ä ¨ ¸À¼¡‡ ± í Ì §Á
Ì òÐ í ..... Å¡ö §¸¡ÀÁ‡ Ì õ ¸¡ÔÁÉø
-¸ ñ Ï º¡Á‡Âõ

þÕÁ‡§Â Å¡¾Óõ À‡ò¾Óõ ÜÊø
..... ¯ ñ ¼ ¯¼ø ¸¡ó¾‡ Î õ
..... ° §½¡ Î ¯È‡ ï º‡ þ É‡ì Ì õ
§¿÷ó¾‡ Î §Á¸õ ¯û ¦Ç§¾¡ ý È‡§Â ¦À¡Õó¾‡ ¦ÁöÂ‡ø
- §¾Ã ý ÁÕòÐÅ À¡Ã¾õ

¯Ú¾‡Ô ûÇ À‡ò¾ÁÐ §¾¡ ý È‡ø ¦ÅôÒ
ÅÇ÷§º¡ ¨ ¸ ÂÆ ¦Ä¡‡× ¸¡ó¾ø

- º¾¸ ¿¡Ê

Ü È¼§Å À¢ò¾ÁÐ Á¢È¢üÈ¡ É¡ø
¦¸¡ Î í ¸¡ó¾Ö¼ý .....
.....¿¡ÅÈðº¢
- « ¸ò¾Â÷ ¿¡Ê

¾¡ý ±ý È À¢ò¾ Á¢È¢ø º¼õ ±øÄ¡ í ¸¡ó¾ø ¸¡ Ï õ

- ¸¡Å¢Âò¾¢ý ¿¡Ê

À¢ò¾ò¾¢ø À¢ò¾Á¡ ¸¢ø
« ¸ò¾Â¡ ÔÄÕ§Á É¢
ÅüÈ¢§Â ¦Å Ù òÐ ì ¸¡Äõ
- « ¸ò¾Â÷ ¿¡Ê

¾ ¨ ÆôÀ¡ É À¢ò¾ò¾¢Ö ðÊ í ¦¸¡ ñ ¼¡ø
..... ¦Â¡¢× ¾¡ ¸õ
- §¿¡ö ¿¡¼ø §¿¡ö Ó¾ø ¿¡¼ø

LINE OF TREATMENT:

1. Since VATHAKARSANAM is a Vatha disease, purgative is given to balance the Vatham.

"tpNurdj;jhy; thjk; jhOk;";

Agasthiyar kuzhambu - 60 mgs early morning with chukka kudineer30ml.

2.MEDICINE:

KARUNGALI VER KUDINEER 30 ml twice a day (before food)

3.DURATION OF TREATMENT:

45 DAYS

4.PATHIYAM AND APATHIYAM:

DIET CHART & GENERAL FOOT CARE ADVICE

ÁÐ§Á¸ ¯½ × Ó ¨È¸û:

¸¡ ¨Ä 6.30 : ¸¡À¢/ Ë/À¡ø (º÷ì ¸¨Ã þøÄ¡Áø)

¸¡ ¨Ä 8.30 : þðÄ¢/§¾¡ ¨º/þÊÂ¡ôÀõ/¦À¡ í ¸ø/
¯ôÒÁ¡/ºôÀ¡¾¢/§¸úÅÃÌ Ü ú/º¡õÀ¡÷/
ºðÉ¢(§¾í ¸¡ö ¾Å¢Ã)/ Óð ¨¼ ¦Åû ¨Ç Áð Îõ.

¸¡ ¨Ä 10.30 : §Á¡÷/ ¸¡ö¸È¢ Ýô/ ¦Åû Ç¡¢ ¸¡ö/¾ì ¸¡Ç¢ Ýô

Á¾¢Âõ 1.30 : º¡¾õ/ ºôÀ¡¾¢/ À¢¦Ãª ý ¦Ã¡ðÊ/ §¸úÅÃÌ ¸Ä¢
º¡õÀ¡÷/ÀÕôÒ/ Á¢ý/ ¸¡¨Ã/¸¡ö¸È¢ ¦À¡È¢Âø/
¾Â¢÷/§Á¡÷

Á¡ ¨ Ä 4.00 : ¸¡À‰/Ë/À¡ø
Í ñ ¼ø/À‰Š ¸ð/º¡ ý Å‰Š/§¸¡Ð ¨ Á À‰Ãð.

þÃ× 7.30 : ºôÀ¡ò¾/þ ðÄ‰/§¾¡ ¨ º/þÊÂ¡ôÀõ/º¡õÀ¡÷/
ÀÕôÒ/§¸¡Æ‰/Á‰ý/¸¡ö¸È‰/
Ãºõ/§Á¡÷/¾Â‰÷/ÀÆõ

þÃ× 9.30 : À¡ø(º÷ì¸¨Ã þøÄ¡Áø)

GENERAL ADVICE:

- Control the blood sugar level
- Follow the diabetic diet advice
- Follow the foot care advice
- Do routine check up like nerve conduction study, Biothesimetry, mono filament test, HbA1C
- Regular check up’s for renal function, eye fundus examination, liver function test

FOOT CARE ADVICE:

- ¦ºöÂ §Å ñ ÊÂ¨ Å:

1.ÁÕòÐÅ¨Ã¯¯¡Â¸¡Ä þ¨¼¦ÅÇ¸Çø ºó¾òÐ¸¡ø¸Ç¸ý À¡¸§º¡¾¨ É¸¨ Ç §Áü¦¸¡ûÇ §Å ñ Î õ.

2.¸¡ø¸¨Ç Íò¾Á¡¸¸ØÅ §Å ñ Î õ.«Åü¨È¿ýÈ¡¸¯Ä÷ò¾§Å ñ Î õ.

3.À¡¾í¸Çø¦ÅÊôÒ¸û,¦¸¡ôÒÇí¸û,¿¸È Á¡ÚÀ¡Î¸û,Å¸ì¸õ ²§¾Ï õ¯ûÇÉÅ¡±É «Êì¸ÊÀ¡¸§º¡¾¸ì¸×õ.

4.ÌÇ¸ìÌõ¾ñ½¦¸ý¦ÅôÀ¿¸¨Ä¨Â¨¸ ãðÊ¨Éì ¦¸¡ñÎ À¡¸§º¡¾¸ì¸×õ.

5.¸¡ø¸¨Ç ±ô§À¡Ðõ ®ÃôÀ¨ºÔ¼ý ¨ÅòÐì¦¸¡ûÇ×õ.

6.¦À¡Õò¾Á¡Éì ¸¡Ä½¸¸¨Ç ÀÂýÀÎò¾×õ.

- ¦ºöÂì Ü¼¡¾¨Å:

1.¦À¡Õó¾¡¾¸¡Ä½¸¸¨Ç «½¸¾ø Ü¼¡Ð.

2. À¡¾í¸¨Ç¿¦ñ¼§¿Ãõ¾ñ½¦¸ø ãú¸¨Åò¾ø Ü¼¡Ð

3.¦ÅüÚì¸¡ø¸Ç¸ø ¿¼ò¾ø Ü¼¡Ð.

4.¸¡ø¸Ç¸ø ²§¾Ïõ¦¸¡ôÒÇõ þÕôÀ¸ý «¨¾¯¨¼ò¾ø Ü¼¡Ð

5.«¾¸¦ÅôÀÁ¡É þ¼í¸Ç¸ø «ÁÕ¾ø Ü¼¡Ð.

6.Ü÷¨ÁÂ¡É¦À¡Õð¸Ç¡ø¿¸ò¨¾¿Õì Ì¾ø Ü¼¡Ð.

7.¸¡Ä¡½¸ÁüÚõ¾Êò¾§¾¡ø þÅü¨È¾¡í¸Ç¡¸§Å¿¦ì Ì¾ø Ü¼¡Ð.

8.ò¨¸ôÀ¸Êò¾¨Äò¾Å¸÷ì¸×õ.²¦É É¸ø ò¨¸ À¸Êò¾ø ¸¡ø¸Ù ì Ì ¦ºøÖõ þÃò¾µð¼ò¨¾ Ì¨Èì Ì õ.

YOGA

- **Yoga practice**

Yogic Physical exercise makes the muscles healthy and strong. It also tones up all the involuntary organs of the body which are concerned with the processes as digestion, evacation, circulation, respiration and secretion and through them, the autonomic nervous system which regulates their activities.

-Yogic Aganas for health & Vigour.

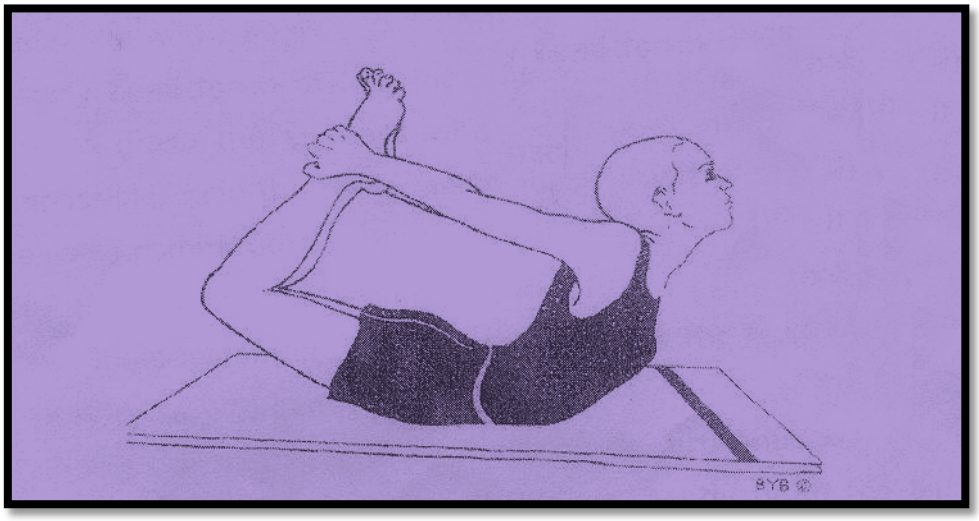
Pranayanam (breathing excersice)

Breathing is regulated with inspiration, expiration and retention of air in the ratio of 1 : 2 : 4

On practicing pranayamam regularly, supply adequate oxygen to nerves cells. The cells of the brain and spinal cord consume much more oxygen. It makes the mind alert and improves concentration.

V.G. Rali

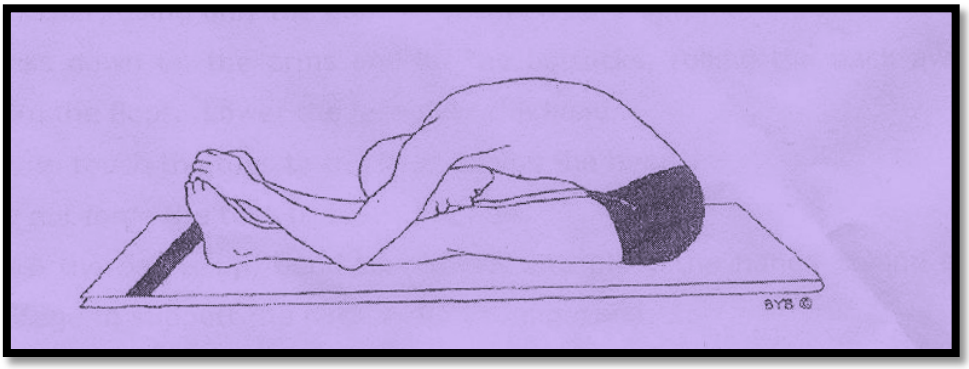
Specific Asanas for Diabetes – Dhanurasana



Dhanurasana (bow pose)

- ❖ Lie flat on the Abdomen with the legs and feet together and the arms and hands beside the body.
- ❖ Bend the knees and bring the heels close to the buttocks.
- ❖ Clasp the hands around the ankles.

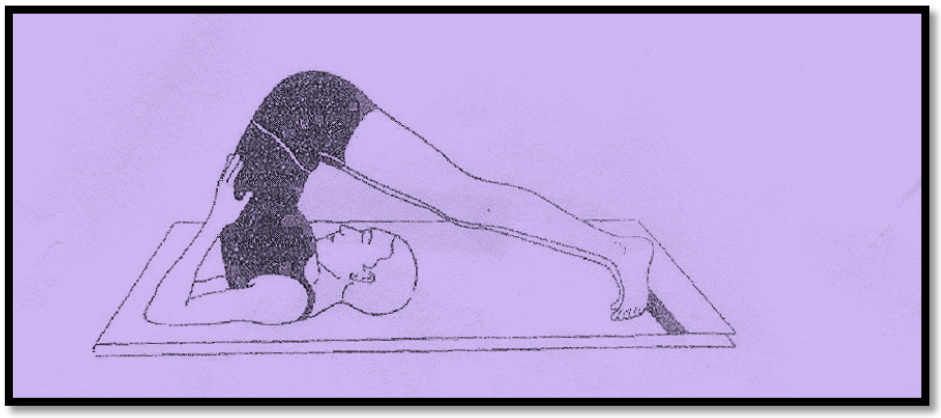
- Place the chin on the floor.
- This is the starting position.
- Tightens the leg muscles and push the feet away from the body. Arch the back, lifting the thighs, chest and head together.
- Keep the arms straight.
- In the final position the head is tilted back and the abdomen supports the entire body on the floor. The only muscular contraction is in the legs; the back and arms remain relaxed.
- Hold the final position for as long as it is comfortable and then slowly relaxing the leg muscles, lower the legs, chest and head to the starting position
- Release the pose and relax in the prone position until the respiration returns to normal.
- **Benefits :** The pancreas and adrenal glands are toned balancing the secretions. It is recommended in Yoga therapy for diabetes.
- **Pachimottanasana (back stretching pose)**



- Sit on the floor with the legs outstretched, feet together and hands on the knees.
- This is the starting position.
- Relax the whole body
- Slowly bend forward from the hips, sliding the hands down the legs. Try to grasp the big toes with the fingers and thumbs. If this is impossible, hold the heels, ankles or any part of the legs that can be reached comfortably.
- Move slowly without forcing or jerking.

- Hold the position for a few seconds. Relax the back and leg muscles allowing them to gently stretch.
- Keeping the legs straight and utilizing the arm muscles, not the back muscles, begin to bend the elbows and gently bring the trunk down towards the legs, maintaining a firm grip on the toes, feet or legs.
- Try to touch the knees with the forehead. Do not strain.
- This is the final position.
- Hold the position for as long as is comfortable and relax.
- Slowly return to the starting position.
- **Benefits :** It tones and massages the entire abdominal and pelvic region including the liver, pancrease, Spleen, Kidneys and adrenal glands.
- **Ardha Matsyendrasana (half spinal twist)**
- Sit with the legs stretched out in front of the body.
- Bend the right leg and place the right foot flat on the foot on the outside of the left knee.
- The toes of the right foot should face forward.
- Bend the left leg and bring the foot around to the right buttock. The outside edge of the foot should be in contact with floor.
- Pass the left arm through the space between the chest and the right knee, and place it agains the outside of the right leg.
- Hold the right foot or ankle with the left hand, so that the right knee is close to the left armpit.
- Sit up as straight as possible.
- Raise the right arm in front of the body and gaze at the fingertips.
- Slowly twist to the right, simultaneously moving the arm, trunk and head.
- Use the left arm as a lever against the right leg to twist the trunk as far as possible without using the back muscles.
- Follow the tips of the fingers of the right hand with the gaze and look over the right shoulder.
- Do not strain the back.

- Bend the right elbow and place the arm around the back of the waist. The back of the right hand should wrap around the left side of the waist.
- Alternatively, it can be placed as high as possible between the shoulder blades with the fingers pointing up. This arm position enforces the straightness of the spine.
- Reverse the movements to come out of the posture and repeat on the other side.
- **Halasana (plough pose)**



- Lie flat on the back with the legs and feet together. Place the arms beside the body with the palms facing down.
- Relax the whole body.
- Raise both legs to the vertical position, keeping them straight and together, using only the abdominal muscles.
- Press down on the arms and lift the buttocks, rolling the back away from the floor. Lower the legs over the head.
- Do not force the toes to touch the floor.
- Turn the palms up, bend the elbows and place the hands behind the ribcage to support the back as in sarvangasana.
- Relax and hold the final pose for as long as is comfortable.
- Return to the starting position by lowering the arms with the plams facing down, then slowly lower the back and buttocks to the floor.
- Raise the legs to the vertical position. Using the abdominal muscles, lower the legs to the starting position, keeping the knees straight

- **Benefits :** It promotes the production of insulin by the pancreas. It boosts the immune system.
- **General Asanas :**
- Surya Namaskara
- Savasana
- Theraiyar clearly explains mild sunrays are much beneficial to our body to prevent diseases.

Savasanam

- By doing savasanam, the whole body is relaxed and rejunuvate the body.
- This Asana being practiced in the world for relaxation techniques.
- Effect of Yoga Asanas on nerve conduction in Type II Diabetes.
- Yoga asanas included "Suryanamskar, Konasan, Padmasan pranayam,

Paschimottansan, Ardhmatsyendrasan, Shavasan. The yoga exercises were performed for 30-40 minutes every day 40 days in above sequence. The subjects were prescribed certain medicines and diet.

Yoga asanas have a beneficial effect on glycaemic control and improve nerve function in mild to moderate type II diabetes with such clinical neuropathy.

-Indian Journal physiology & Pharmacology 2002 : 46 (3)

Varun Malhotra etal

Stress Management

Diagnosis of diabetes mellitus is a stressful situation in life of an individual and appropriate management requires a holistic approach that includes behavioral modification to develop positive attitude and healthy life style. A satisfactory treatment plan should include special attention to person with diabetes, quality of life, coping skills, optimal family support and a healthy workplace environment. Appropriate support and counseling is an essential component of the management at the time of diagnosis and throughout life.

6. **PREVENTION:**

- Balanced diet
- Regular exercise
- Suriya namesakar
- Oil bath twice in a week
- Avoid Junk foods & sweets
- Avoid tobacco
- Avoid Contraceptives

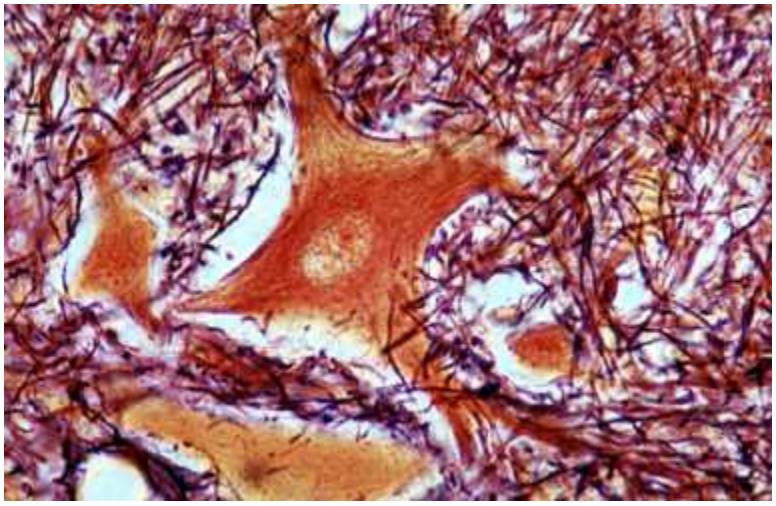
MODERN ASPECTS

MODERN ASPECT

The Neuron

Neurons

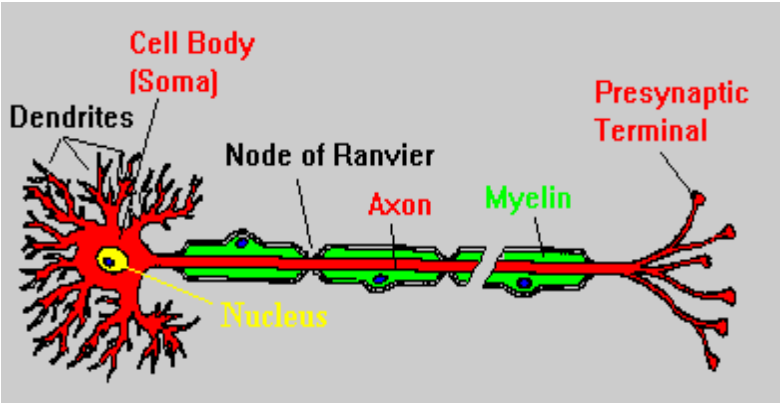
It is clear that most of what we think of as our mental life involves the activities of the nervous system, especially the brain. This nervous system is composed of billions of



cells, the most essential being the nerve cells or neurons. There are estimated to be as many as 100 billion neurons in our nervous systemspinal cord neuron

A typical neuron has all the parts that any cell would have, and a few specialized structures that set it apart. The main portion of the cell is called the **soma** or **cell body**. It contains the **nucleus**, which in turn contains the genetic material in the form of chromosomes.

Neurons have a large number of extensions called **dendrites**. They often look likes branches or spikes extending out from the cell body. It is primarily the surfaces of the dendrites that receive chemical messages from other neurons.

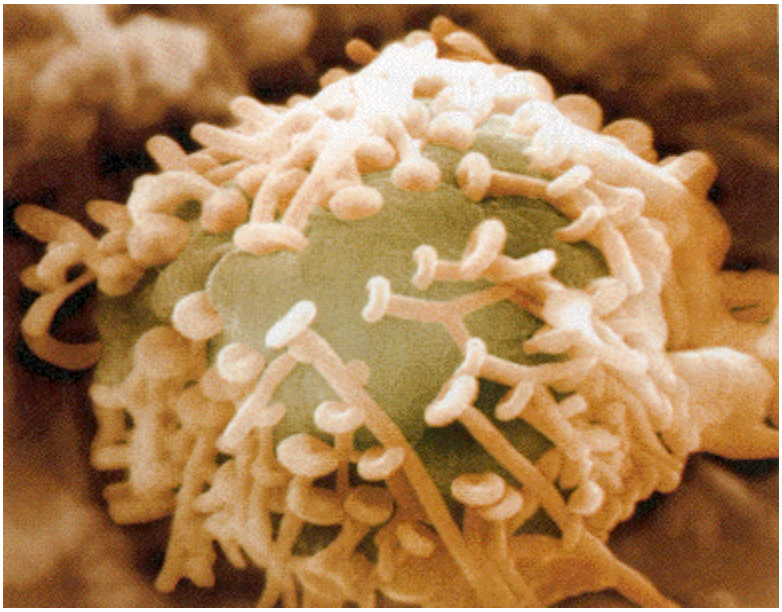


One extension is different from all the others, and is called the **axon**. Although in some neurons, it is hard to distinguish from

the dendrites, in others it is easily distinguished by its length. The purpose of the axon is to transmit an electro-chemical signal to other neurons, sometimes over a considerable distance. In the neurons that make up the nerves running from the spinal cord to your toes, the axons can be as long as three feet.

Longer axons are usually covered with a **myelin sheath**, a series of fatty cells which have wrapped around an axon many times. These make the axon look like a necklace of sausage-shaped beads. They serve a similar function as the insulation around electrical wire.

At the very end of the axon is the **axon ending**, which goes by a variety of names such as the bouton, the synaptic knob, the axon foot, and so on It is there that the electro-chemical signal that has travelled the length of the axon is converted into a chemical message that travels to the next neuron.

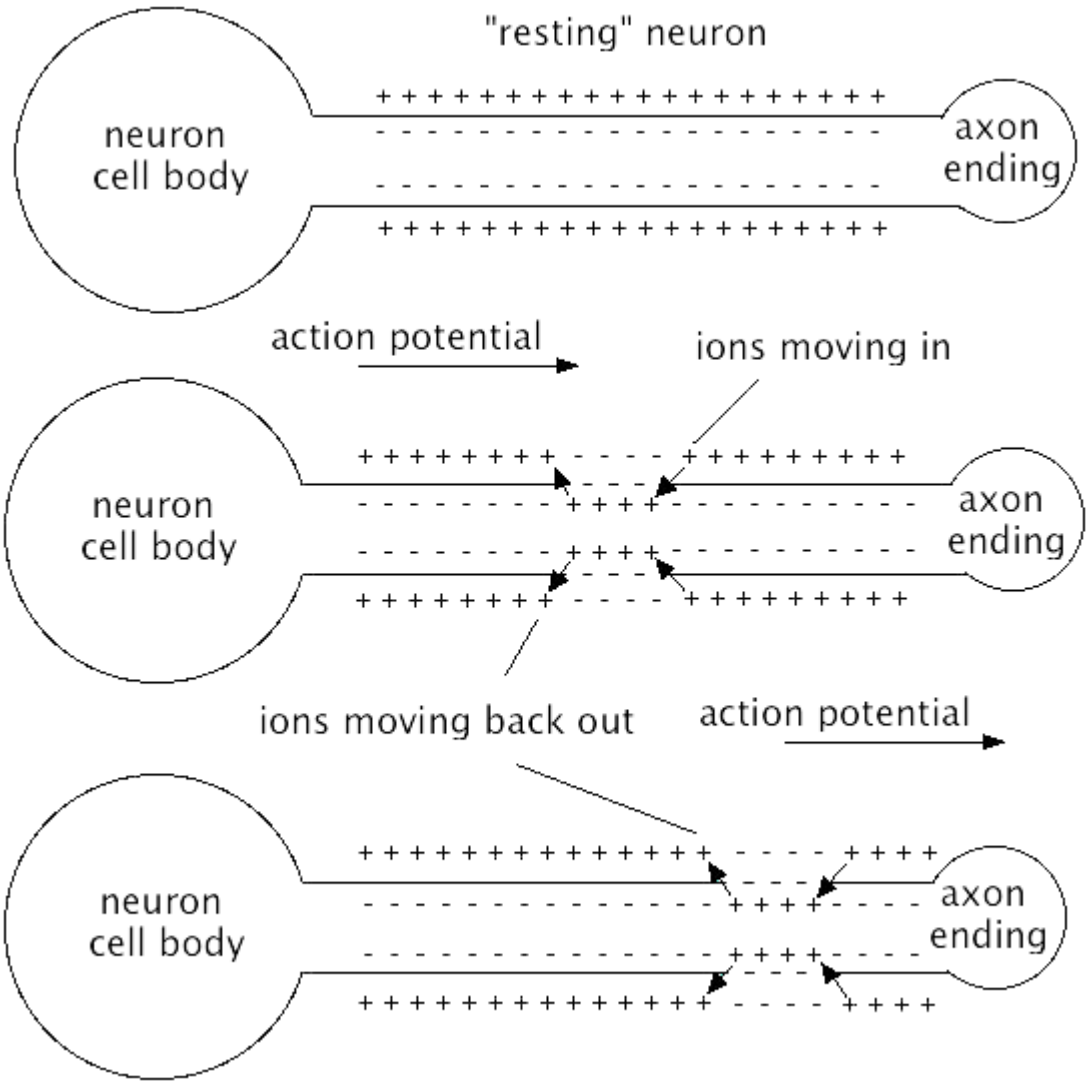


axon endings

Between the axon ending and the dendrite of the next neuron is a very tiny gap called the **synapse** (or synaptic gap, or synaptic cleft), which we will discuss in a little bit. For every neuron, there are between 1000 and 10,000 synapses.

When chemicals contact the surface of a neuron, they change the balance of **ions** (electrically charged atoms) between the inside and outside of the cell membrane. When this change reaches a threshold level, this effect runs across the cell's membrane to the axon. When it reaches the axon, it initiates the **action potential**, which is a rapidly moving exchange of ions.

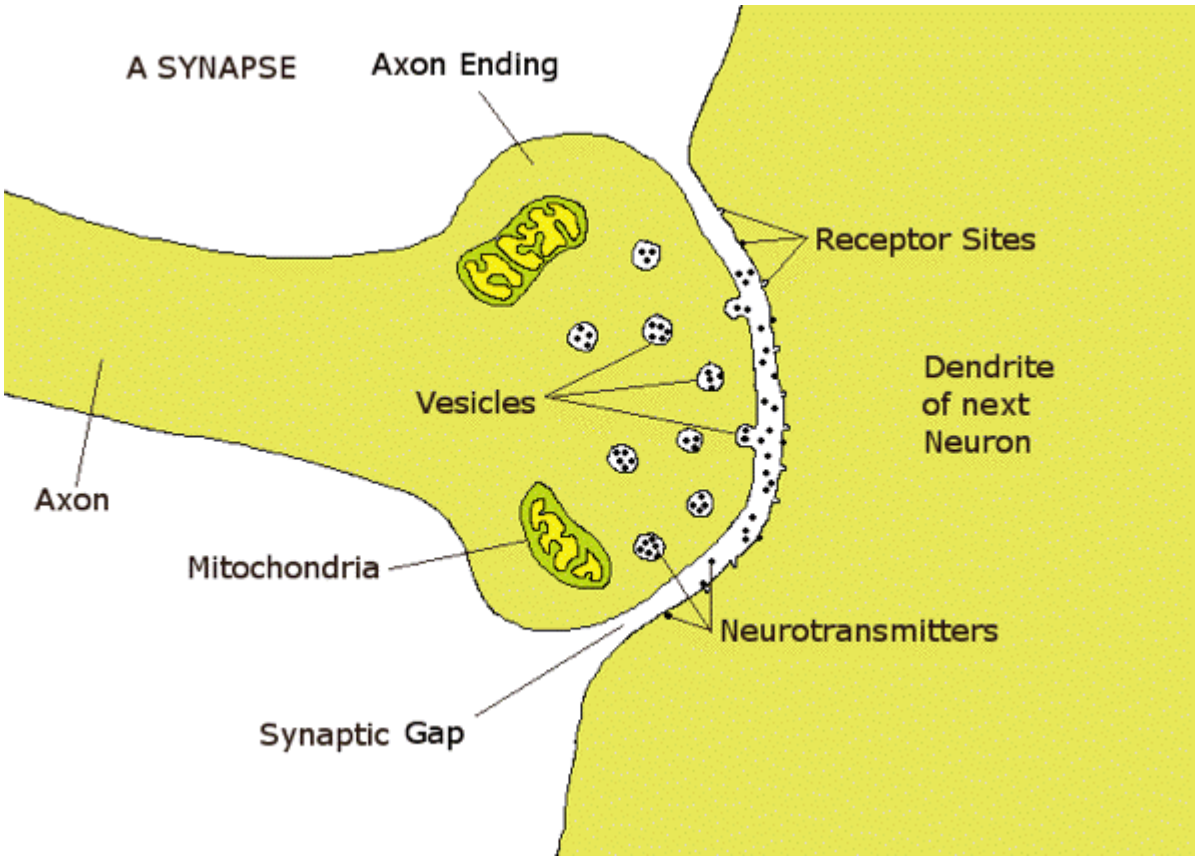
The surface of the axon contains hundreds of thousands of miniscule mechanisms called **ion channels**. When the charge enters the axon, the ion channels at the base of the axon allow positively charged ions to enter the axon, changing the electrical balance between inside and outside. This causes the next group of ion channels to do the same, while other channels return positive ions to the outside, and so on all the way down the axon.



In this little diagram, the red represents the positive ions going into the axon, while the orange represents positive ions going out. The action potential travels at a rate of 1.2 to 250 miles per hour

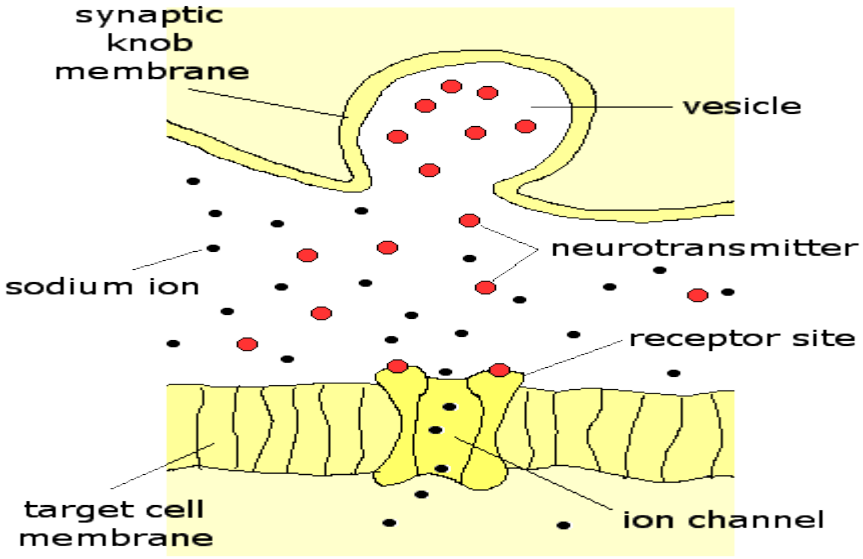
The synapse:

When the action potential reaches the axon ending, it causes tiny bubbles of chemicals called **vesicles** to release their contents into the synaptic gap. These chemicals are called **neurotransmitters**. These sail across the gap to the next neuron, where they find special places on the cell membrane of the next neuron called **receptor sites**.



The neurotransmitter acts like a little key, and the receptor site like a little lock. When they meet, they open a passage way for ions, which then change the balance of ions on the outside and the inside of the next neuron. And the whole process starts all over again.

While most neurotransmitters are **excitatory** -- i.e. they excite the next neuron -- there are also **inhibitory** neurotransmitters. These make it more difficult for the excitatory neurotransmitters to have their effect.



Types of Neurons (Nerve Cells)

While there are many different kinds of neurons, there are three broad categories based on function:

1. **Sensory neurons** are sensitive to various non-neural stimuli. There are sensory neurons in the skin, muscles, joints, and organs that indicate pressure, temperature, and pain. There are more specialized neurons in the nose and tongue that are sensitive to the molecular shapes we perceive as tastes and smells. Neurons in the inner ear are sensitive to vibration, and provide us with information about sound. And the rods and cones of the retina are sensitive to light, and allow us to see.

2. **Motor neurons** are able to stimulate muscle cells throughout the body, including

the muscles of the heart, diaphragm, intestines, bladder, and glands.

3. **Interneurons** are the neurons that provide connections between sensory and motor neurons, as well as between themselves. The neurons of the central nervous system, including the brain, are all interneurons.

Most neurons are collected into "packages" of one sort or another, sometimes visible to the naked eye. A clump of neuron cell bodies, for example, is called a **ganglion** (plural: **ganglia**) or a **nucleus** (plural: **nuclei**). A fiber made up of many axons is called a **nerve**. In the brain and spinal cord, areas that are mostly axons are called **white matter**, and it is possible to differentiate **pathways** or **tracts** of these axons. Areas that include large number of cell bodies are called **gray matter**.

One way to classify neurons is by the number of extensions that extend from the neuron's cell body (soma).

Bipolar neurons have two processes extending from the cell body (examples: retinal cells, olfactory epithelium cells).

Pseudounipolar cells (example: dorsal root ganglion cells). Actually, these cells have 2 axons rather than an axon and dendrite. One axon extends centrally toward the spinal cord, the other axon extends toward the skin or muscle.

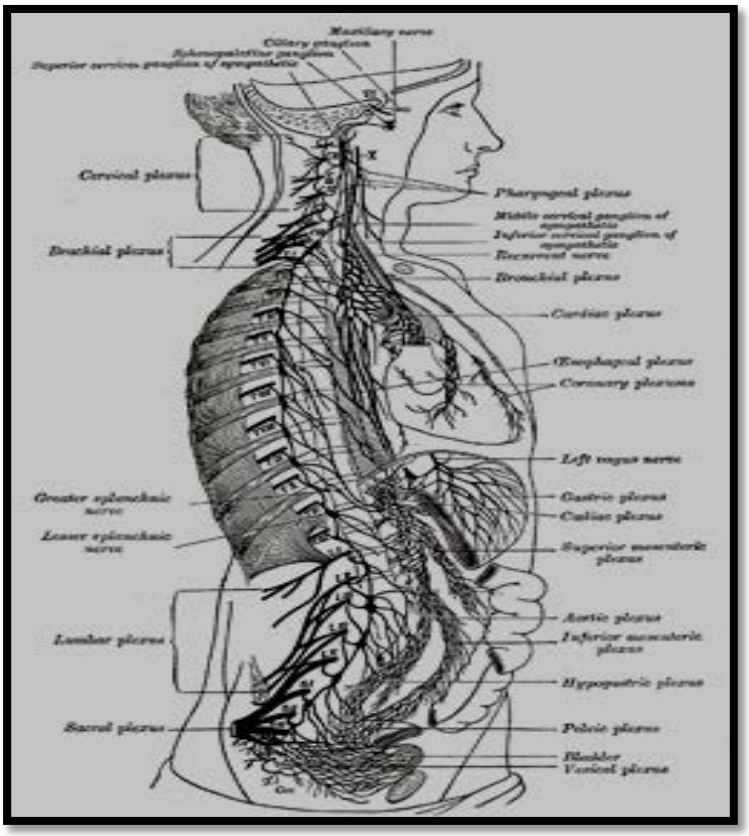
Multipolar neurons have many processes that extend from the cell body. However, each neuron has only one axon (examples: spinal motor neurons, pyramidal neurons, Purkinje cells).

Neurons can also be classified by the direction that they send information.

- **Sensory (or afferent) neurons:** send information from sensory receptors (e.g., in skin, eyes, nose, tongue, ears) **TOWARD** the central nervous system.
- **Motor (or efferent) neurons:** send information **AWAY** from the central nervous system to muscles or glands.
- **Interneurons:** send information between sensory neurons and motor neurons. Most interneurons are located in the central nervous system.

The Sympathetic Nerves

The **sympathetic nervous system** innervates all the smooth muscles and the various glands of the body, and the striated muscle of the heart. The efferent sympathetic fibers which leave the central nervous system in connection with certain of the cranial and spinal nerves all end in sympathetic ganglia and are known as **preganglionic fibers.** From these ganglia postganglionic fibers arise and conduct impulses to the different organs. In addition, afferent or sensory fibers connect many of these structures with the central nervous system.



The right sympathetic chain and its connections with the thoracic, abdominal, and pelvic plexuses. (After Schwalbe.)

The peripheral portion of the sympathetic nervous system is characterized by the presence of numerous ganglia and complicated plexuses. These ganglia are connected with the central nervous system by three groups of sympathetic efferent or preganglionic fibers, *i. e.*, the **cranial,** the **thoracolumbar,** and the **sacral.** These outflows of sympathetic fibers are separated by intervals where no connections exist. The cranial and sacral sympathetics are often grouped together owing to the resemblance between the reactions produced by stimulating them and by the effects of certain drugs. Acetyl-choline, for example, when injected intravenously in very small doses, produces the same effect as the stimulation of the cranial or sacral sympathetics, while the introduction of adrenalin produces the same effect as the stimulation of the thoracolumbar sympathetics. Much of our present knowledge of the sympathetic nervous system has been acquired through the application of various

drugs, especially nicotine which paralyzes the connections or synapses between the preganglionic and postganglionic fibers of the sympathetic nerves. When it is injected into the general circulation all such synapses are paralyzed; when it is applied locally on a ganglion only the synapses occurring in that particular ganglion are paralyzed.

Langley, who has contributed greatly to our knowledge, adopted a terminology somewhat different from that used here and still different from that used by the pharmacologists. This has led to considerable confusion, as shown by the arrangement of the terms in the following columns. Gaskell has used the term involuntary nervous system.

Gray.	Langley.	Meyer and Gottlieb.
Sympathetic nervous system.	Autonomic nervous system.	Vegetative nervous system.
Cranio-sacral sympathetics.	Parasympathetics.	Autonomic.
Oculomotor sympathetics.	Tectal autonomics.	Cranial autonomics.
Facial Fsympathetics.		
Glossopharyngeal sympathetics.	Bulbar autonomics.	
Vagal sympathetics.		
Sacral sympathetics.	Sacral autonomics.	Sacral autonomics.
Thoracolumbar sympathetics.	Sympathetic.	Sympathetic.
	Thoracic autonomic.	
Enteric.	Enteric.	Enteric.

The Cranial Sympathetics:

The **cranial sympathetics** include sympathetic efferent fibers in the oculomotor, facial, glossopharyngeal and vagus nerves, as well as sympathetic afferent in the last three nerves.

The **Sympathetic Efferent Fibers of the Oculomotor Nerve** probably arise from cells in the anterior part of the oculomotor nucleus which is located in the tegmentum of the mid-brain. These preganglionic fibers run with the third nerve into the orbit and pass to the ciliary ganglion where they terminate by forming synapses with sympathetic motor neurons whose axons, postganglionic fibers, proceed as the short ciliary nerves to the eyeball. Here they supply motor fibers to the Ciliaris muscle and the Sphincter pupillæ muscle. So far as known there are no sympathetic afferent fibers connected with the nerve.

The **Sympathetic Efferent Fibers of the Facial Nerve** are supposed to arise from the small cells of the facial nucleus. According to some authors the fibers to the salivary glands arise from a special nucleus, the superior salivatory nucleus, consisting of cells scattered in the reticular formation, dorso-medial to the facial nucleus. These preganglionic fibers are distributed partly through the chorda tympani and lingual nerves to the submaxillary ganglion where they terminate about the cell bodies of neurons whose axons as postganglionic fibers conduct secretory and vasodilotar impulses to the submaxillary and sublingual glands. Other preganglionic fibers of the facial nerve pass via the great superficial petrosal nerve to the sphenopalatine ganglion where they form synapses with neurons whose postganglionic fibers are distributed with the superior maxillary nerve as vasodilator and secretory fibers to the mucous membrane of the nose, soft palate, tonsils, uvula, roof of the mouth, upper lips and gums, parotid and orbital glands.

There are supposed to be a few sympathetic afferent fibers connected with the facial nerve, whose cell bodies lie in the geniculate ganglion, but very little is known about them.

The **Sympathetic Afferent Fibers of the Glossopharyngeal Nerve** are supposed to arise either in the dorsal nucleus (nucleus ala cinerea) or in a distinct nucleus, the inferior salivatory nucleus, situated near the dorsal nucleus. These preganglionic fibers pass into the tympanic branch of the glossopharyngeal and then with the small superficial petrosal nerve to the otic ganglion. Postganglionic fibers, vasodilator and secretory fibers, are distributed to the parotid gland, to the mucous

membrane and its glands on the tongue, the floor of the mouth, and the lower gums.

Sympathetic Afferent Fibers, whose cells of origin lie in the superior or inferior ganglion of the trunk, are supposed to terminate in the dorsal nucleus. Very little is known of the peripheral distribution of these fibers.

The **Sympathetic Efferent Fibers of the Vagus Nerve** are supposed to arise in the dorsal nucleus (nucleus ala cinerea). These preganglionic fibers are all supposed to end in sympathetic ganglia situated in or near the organs supplied by the vagus sympathetics. The inhibitory fibers to the heart probably terminate in the small ganglia of the heart wall especially the atrium, from which inhibitory postganglionic fibers are distributed to the musculature. The preganglionic motor fibers to the esophagus, the stomach, the small intestine, and the greater part of the large intestine are supposed to terminate in the plexuses of Auerbach, from which postganglionic fibers are distributed to the smooth muscles of these organs. Other fibers pass to the smooth muscles of the bronchial tree and to the gall-bladder and its ducts. In addition the vagus is believed to contain secretory fibers to the stomach and pancreas. It probably contains many other efferent fibers than those enumerated above.

Sympathetic Afferent Fibers of the Vagus, whose cells of origin lie in the jugular ganglion or the ganglion nodosum, probably terminate in the dorsal nucleus of the medulla oblongata or according to some authors in the nucleus of the tractus solitarius. Peripherally the fibers are supposed to be distributed to the various organs supplied by the sympathetic efferent fibers.

The Sacral Sympathetics

The **Sacral Sympathetic Efferent Fibers** leave the spinal cord with the anterior roots of the second, third and fourth sacral nerves. These small medullated preganglionic fibers are collected together in the pelvis into the nervus erigentes or pelvic nerve which proceeds to the hypogastric or pelvic plexuses from which postganglionic fibers are distributed to the pelvic viscera. Motor fibers pass to the smooth muscle of the descending colon, rectum, anus and bladder. Vasodilators

are distributed to these organs and to the external genitalia, while inhibitory fibers probably pass to the smooth muscles of the external genitalia. **Afferent sympathetic fibers** conduct impulses from the pelvic viscera to the second, third and fourth sacral nerves. Their cells of origin lie in the spinal ganglia.

The Thoracolumbar Sympathetics:

The **thoracolumbar sympathetic fibers** arise from the dorso-lateral region of the anterior column of the gray matter of the spinal cord and pass with the anterior roots of all the thoracic and the upper two or three lumbar spinal nerves. These preganglionic fibers enter the white rami communicantes and proceed to the sympathetic trunk where many of them end in its ganglia, others pass to the prevertebral plexuses and terminate in its collateral ganglia. The postganglionic fibers have a wide distribution. The **vasoconstrictor fibers** to the bloodvessels of the skin of the trunk and limbs, for example, leave the spinal cord as preganglionic fibers in all the thoracic and the upper two or three lumbar spinal nerves and terminate in the ganglia of the sympathetic trunk, either in the ganglion directly connected with its ramus or in neighboring ganglia. Postganglionic fibers arise in these ganglia, pass through gray rami communicantes to all the spinal nerves, and are distributed with their cutaneous branches, ultimately leaving these branches to join the small arteries. The postganglionic fibers do not necessarily return to the same spinal nerves which contain the corresponding preganglionic fibers. The vasoconstrictor fibers to the head come from the upper thoracic nerves, the preganglionic fibers end in the superior cervical ganglion. The postganglionic fibers pass through the internal carotid nerve and branch from it to join the sensory branches of the various cranial nerves, especially the trigeminal nerve; other fibers to the deep structures and the salivary glands probably accompany the arteries.

The postganglionic vasoconstrictor fibers to the bloodvessels of the abdominal viscera arise in the prevertebral or collateral ganglia in which terminate

many preganglionic fibers. Vasoconstrictor fibers to the pelvic viscera arise from the inferior mesenteric ganglia.

The pilomotor fibers to the hairs and the motor fibers to the sweat glands apparently have a distribution similar to that of the vasoconstrictors of the skin.

A vasoconstrictor center has been located by the physiologists in the neighborhood of the facial nucleus. Axons from its cells are supposed to descend in the spinal cord to terminate about cell bodies of the preganglionic fibers located in the dorsolateral portion of the anterior column of the thoracic and upper lumbar region.

The motor supply to the dilator pupillæ muscle of the eye comes from preganglionic sympathetic fibers which leave the spinal cord with the anterior roots of the upper thoracic nerves. These fibers pass to the sympathetic trunk through the white rami communicantes and terminate in the superior cervical ganglion. Postganglionic fibers from the superior cervical ganglion pass through the internal carotid nerve and the ophthalmic division of the trigeminal nerve to the orbit where the long ciliary nerves conduct the impulses to the eyeball and the dilator pupillæ muscle. The cell bodies of these preganglionic fibers are connected with fibers which descend from the mid-brain.

Other postganglionic fibers from the superior cervical ganglion are distributed as secretory fibers to the salivary glands, the lacrimal glands and to the small glands of the mucous membrane of the nose, mouth and pharynx.

The thoracic sympathetics supply accelerator nerves to the heart. They are supposed to emerge from the spinal cord in the anterior roots of the upper four or five thoracic nerves and pass with the white rami to the first thoracic ganglion, here some terminate, others pass in the ansa subclavia to the inferior cervical ganglion. The postganglionic fibers pass from these ganglia partly through the ansa subclavia to the heart, on their way they intermingle with sympathetic fibers from the vagus to form the cardiac plexus.

Inhibitory fibers to the smooth musculature of the stomach, the small intestine and most of the large intestine are supposed to emerge in the anterior roots of the lower thoracic and upper lumbar nerves. These fibers pass through the white rami and

sympathetic trunk and are conveyed by the splanchnic nerves to the prevertebral plexus where they terminate in the collateral ganglia. From the celiac and superior mesenteric ganglia postganglionic fibers (inhibitory) are distributed to the stomach, the small intestine and most of the large intestine. Inhibitory fibers to the descending colon, the rectum and Internal sphincter ani are probably postganglionic fibers from the inferior mesenteric ganglion.

The thoracolumbar sympathetics are characterized by the presence of numerous ganglia which may be divided into two groups,**central** and **collateral.**

The **central ganglia** are arranged in two vertical rows, one on either side of the middle line, situated partly in front and partly at the sides of the vertebral column. Each ganglion is joined by intervening nervous cords to adjacent ganglia so that two chains, the**sympathetic trunks,** are formed. The **collateral ganglia** are found in connection with three great **prevertebral plexuses,**placed within the thorax, abdomen, and pelvis respectively.

The **sympathetic trunks** (*truncus sympathicus; gangliated cord*) extend from the base of the skull to the coccyx. The cephalic end of each is continued upward through the carotid canal into the skull, and forms a plexus on the internal carotid artery; the caudal ends of the trunks converge and end in a single ganglion, the **ganglion impar,** placed in front of the coccyx. The ganglia of each trunk are distinguished as **cervical, thoracic, lumbar,** and **sacral** and, except in the neck, they closely correspond in number to the vertebræ. They are arranged thus:

Cervical portion	3 ganglia
Thoracic portion	12 ganglia
Lumbar portion	4 ganglia
Sacral portion	4 or 5 ganglia

In the neck the ganglia lie in front of the transverse processes of the vertebræ; **in the thoracic region** in front of the heads of the ribs; **in the lumbar region** on the sides of the vertebral bodies; and **in the sacral region** in front of the sacrum.

Connections with the Spinal Nerves.—Communications are established between the sympathetic and spinal nerves through what are known as the **gray** and **white rami communicantes** the gray rami convey sympathetic fibers into the spinal nerves and the white rami transmit spinal fibers into the sympathetic. Each spinal nerve receives a gray ramus communicans from the sympathetic trunk, but white rami are not supplied by all the spinal nerves. White rami are derived from the first thoracic to the first lumbar nerves inclusive, while the visceral branches which run from the second, third, and fourth sacral nerves directly to the pelvic plexuses of the sympathetic belong to this category. The fibers which reach the sympathetic through the white rami communicantes are medullated; those which spring from the cells of the sympathetic ganglia are almost entirely non-medullated. .

The **three great gangliated plexuses** (*collateral ganglia*) are situated in front of the vertebral column in the thoracic, abdominal, and pelvic regions, and are named, respectively, the **cardiac,** the **solar** or **epigastric,** and the **hypogastric plexuses.** They consist of collections of nerves and ganglia; the nerves being derived from the sympathetic trunks and from the cerebrospinal nerves. They distribute branches to the viscera.

DIABETIC NEUROPATHY

Diabetic neuropathies are a family of nerve disorders caused by diabetes. People with diabetes can, over time, develop nerve damage throughout the body. Some people with nerve damage have no symptoms. Others may have symptoms such as pain, tingling, or numbness-loss of feeling-in the hands, arms, feet, and legs. Nerve problems can occur in every organ system, including the digestive tract, heart, and sex organs.

About 60 to 70 percent of people with diabetes have some form of neuropathy. People with diabetes can develop nerve problems at any time, but risk rises with age and longer duration of diabetes. The highest rates of neuropathy are among people who have had diabetes for at least 25 years. Diabetic neuropathies also appear to be more common in people who have problems controlling their blood glucose, also called blood sugar.

Epidemiology

A large American study estimated that 47% of patients with diabetes have some peripheral neuropathy. Neuropathy is estimated to be present in 7.5% of patients at the time of diabetes diagnosis. More than half of cases are distal symmetric polyneuropathy. Focal syndromes such as carpal tunnel syndrome (14-30%) radiculopathies/plexopathies, and cranial neuropathies account for the rest. Solid prevalence data for the latter 2 less-common syndromes is lacking.

The wide variability in symmetric diabetic polyneuropathy prevalence data is due to lack of consistent criteria for diagnosis, variable methods of selecting patients for study, and differing assessment techniques. In addition, because many patients with diabetic polyneuropathy are initially asymptomatic, detection is extremely dependent on careful neurologic examination by the primary care clinician. The use of additional diagnostic techniques, such as autonomic or quantitative sensory testing, might result in a higher recorded prevalence.

International statistics

In a cohort of 4400 Belgian patients, Pirart et al found that 7.5% of patients already had neuropathy when diagnosed with diabetes. After 25 years, the number with neuropathy rose to 45%. In the United Kingdom, the prevalence of diabetic neuropathy among the hospital clinic population was noted to be around 29%.

Diabetic neuropathy in racial minorities

No definite racial predilection has been demonstrated for diabetic neuropathy. However, members of minority groups (eg, Hispanics, African Americans) have more secondary complications from diabetic neuropathy, such as lower-extremity amputations, than whites. They also have more hospitalizations for neuropathic complications.

Sex differences in diabetic neuropathy

DM affects men and women with equal frequency. However, male patients with type 2 diabetes may develop diabetic polyneuropathy earlier than female patients, and neuropathic pain causes more morbidity in females than in males.

Diabetic neuropathy and advancing age

Diabetic neuropathy can occur at any age but is more common with increasing age and severity and duration of diabetes

Etiology

Risk factors that are associated with more severe symptoms include the following

- Poor glycemic control
- Advanced age
- Hypertension
- Long duration of DM
- Dyslipidemia
- Smoking
- Heavy alcohol intake
- HLA-DR3/4 phenotype
- Tall height

Development of symptoms depends on many factors, such as total hyperglycemic exposure and other risk factors such as elevated lipids, blood pressure, smoking, increased height, and high exposure to other potentially neurotoxic agents such as ethanol. Genetic factors may also play a role

Peripheral neuropathies have been described in patients with primary DM (types 1 and 2) and in those with secondary diabetes of diverse causes, suggesting a common etiologic mechanism based on chronic hyperglycemia. The contribution of hyperglycemia has received strong support from the Diabetes Control and Complications Trial (DCCT).

SYMPTOMS:

Symptoms depend on the type of neuropathy and which nerves are affected. Some people with nerve damage have no symptoms at all. For others, the first symptom is often numbness, tingling, or pain in the feet. Symptoms are often

minor at first, and because most nerve damage occurs over several years, mild cases may go unnoticed for a long time. Symptoms can involve the sensory, motor, and autonomic-or involuntary-nervous systems. In some people, mainly those with focal neuropathy, the onset of pain may be sudden and severe.

- Burning sensation in plants
- Pain in calf muscles
- Numbness of soles
- Glove and stocking type of anesthesia
- Weakness of lower limbs.

- wasting of the muscles of the feet or hands
- indigestion, nausea, or vomiting
- diarrhea or constipation
- dizziness or faintness due to a drop in blood pressure after standing or sitting up

STAGES

S.NO	STAGES	SYMPTOMS
1.	ACUTE PAINFUL	Burning, shooting stabbing pain, pins and needles, increased at nights absent/reduced reflexes
2.	CHRONIC PAINFUL	Severe symptoms as above (Hyperasthesia common) in poorly controlled DM
3.	PAINLESS WITH COMPLETE/PARTIAL SENSORY LOSS	Numbness/ deadness of feet painless injury, absent of jerks.

Polymotor-Sensory Neuropathy

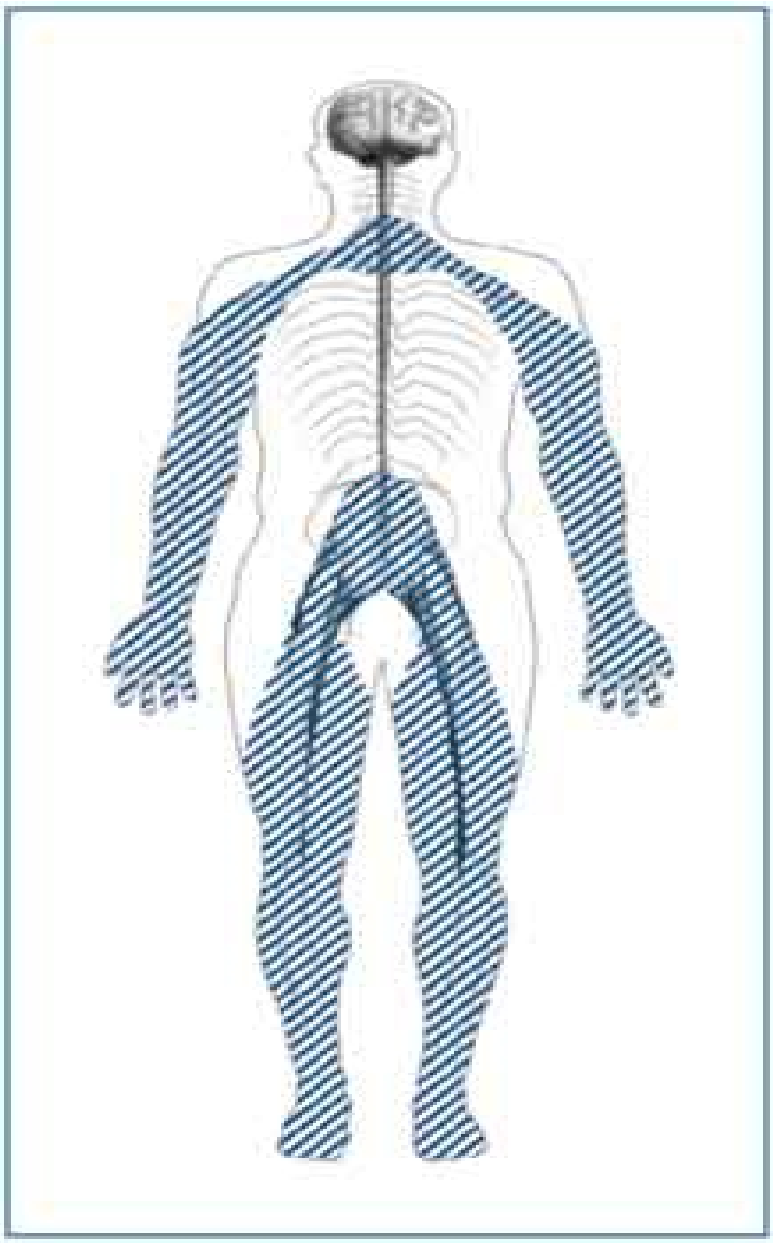
In the early phase of diabetic neuropathy, physical examination may be uninformative. Assessing the adequacy of perfusion is critical and easily accomplished by inspecting the toenails for capillary perfusion and palpating the tibialis posterior and dorsalis pedis arteries. As noted, a dry, cool foot with thickened or thin subcutaneous tissue and nonelastic skin is demonstrating microangiopathy, despite a palpable dorsalis pedis, indicating a likely positive result in decreased deep tendon reflexes at the ankle, vibratory sensation, position sense, hot-cold discrimination, or fine touch with monofilament test. Inability to perceive the monofilament indicates advanced neuropathy that would endanger the foot if injured. Peripheral neuropathic symptoms may also result from hyperglycemia-mediated nerve injury with no clinical evidence of loss of capillary density. Over time, paresthesias and burning pains usually improve, unless functional factors (endogenous or related to narcotic therapy) prolong or confound the symptomatology.

Loss of the ankle reflex with sensory abnormalities indicates that the classic diabetic polyneuropathy is fully defined, with probable coexisting microangiopathy in the foot. Preventive foot care needs to be practiced to prevent skin breakdown. Patients should never walk barefooted because sharp objects penetrating the diabetic foot are often not perceived, leading to foot-threatening infections.

Mononeuropathies

An individual nerve may be affected, such as the peroneal, resulting in footdrop, the seventh cranial nerve causing Bell’s palsy, or the extraocular nerves causing strabismus and diplopias. Similar to other causes of Bell’s palsy, the pathogenesis of these large-nerve injuries is thought to result from vascular injury; the paralysis is usually self-limiting and spontaneously improves over several months. In general, all these neuropathies have become less common as average HbA1c levels have fallen from above the 9% range over the last 10 years.

Occasionally, a diabetic mononeuritis with a localized region of pain can be misdiagnosed clinically as a mechanically mediated neuritis, such as a herniated intravertebral disk. If the appropriate imaging studies fail to demonstrate a mechanical etiology, a presumptive diagnosis of diabetes neuritis should be made and treatment aimed at glycemic control with physical therapy. On the other hand, diabetic neuritis of this type with constant pain may be initiated by mechanical factors. For example, a diabetic patient may acutely injure spinal nerves with a lifting maneuver, but this “neuromuscular pain” may become a continuing neuropathic pain as diabetic nerve fibers fail to heal in the presence of marked hyperglycemia. With no other etiologies evident, these patients eventually improve with more effective diabetic management.



DISTRIBUTION OF PAIN

PATHOLOGY

The factors leading to the development of diabetic neuropathy are not understood completely, and multiple hypotheses have been advanced.[1] It is generally accepted to be a multifactorial process. Development of symptoms depends on many factors, such as total hyperglycemic exposure and other risk factors such as elevated lipids, blood pressure, smoking, increased height, and high exposure to other potentially neurotoxic agents such as ethanol. Genetic factors may also play a role. Important contributing biochemical mechanisms in the development of the more common symmetrical forms of diabetic polyneuropathy likely include the polyol pathway, advanced glycation end products, and oxidative stress.

Polyol pathway

Hyperglycemia causes increased levels of intracellular glucose in nerves, leading to saturation of the normal glycolytic pathway. Extra glucose is shunted into the polyol pathway and converted to sorbitol and fructose by the enzymes aldose reductase and sorbitol dehydrogenase. Accumulation of sorbitol and fructose lead to reduced nerve myoinositol, decreased membrane Na^+/K^+ -ATPase activity, impaired axonal transport, and structural breakdown of nerves, causing abnormal action potential propagation. This is the rationale for the use of aldose reductase inhibitors to improve nerve conduction.

Advanced glycation end products

The nonenzymatic reaction of excess glucose with proteins, nucleotides, and lipids results in advanced glycation end products (ACE) that may have a role in disrupting neuronal integrity and repair mechanisms through interference with nerve cell metabolism and axonal transport.

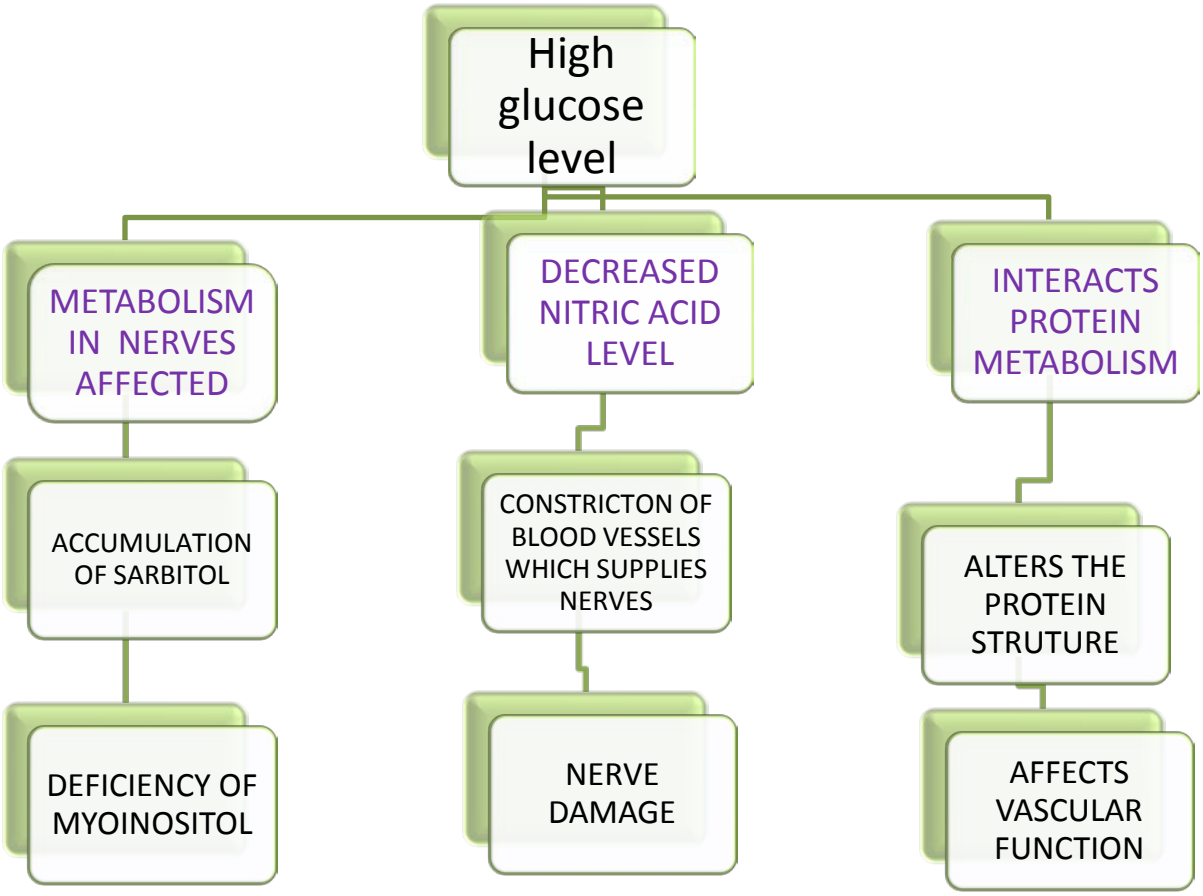
Oxidative stress

The increased production of free radicals in diabetes may be detrimental via several mechanisms that are not fully understood. These include direct damage to blood vessels leading to nerve ischemia and facilitation of AGE reactions. Despite the

incomplete understanding of these processes, use of the antioxidant alpha-lipoic acid may hold promise for improving neuropathic symptoms.

Related contributing factors

Problems that are a consequence of or co-contributors to these disturbed biochemical processes include altered gene expression with altered cellular phenotypes, changes in cell physiology relating to endoskeletal structure or cellular transport, reduction in neurotrophins, and nerve ischemia. Clinical trials of the best-studied neurotrophin, human recombinant nerve growth factor, were disappointing. With future refinements, however, pharmacologic intervention targeting one or more of these mechanisms may prove successful.



INVESTIGATION

NERVE CONDUCTION STUDY &ELECTROMYOGRAPHY:

Nerve conduction velocity (NCV) test--also called a nerve conduction study (NCS)--is a measurement of the speed of conduction of an electrical impulse through a nerve. NCV can determine nerve damage and destruction.During the test, the nerve is stimulated, usually with surface electrode patches attached to the skin. Two electrodes are placed on the skin over the nerve. One electrode stimulates the nerve with a very mild electrical impulse and the other electrode records it. The resulting electrical activity is recorded by another electrode. This is repeated for each nerve being tested.The nerve conduction velocity (speed) is then calculated by measuring the distance between electrodes and the time it takes for electrical impulses to travel between electrodes.

A related procedure that may be performed is electromyography (EMG). An EMG measures the electrical activity in muscles and is often performed at the same time as NCV. Both procedures help to detect the presence, location, and extent of diseases that damage the nerves and muscles.

ELECTRO PHYSIOLOGICAL STUDIES:

In generalized symmetrical neuropathies there is impairment of motor and sensory conduction

BIOTHESIMETRY:

Testing vibration sensation with a biothesiometer - application guidelines:

The biothesiometer has readings from 0 to 50 volts. It can be made to vibrate at increasing intensity by turning a dial.A probe is applied to part of the foot, usually on the big toe.The person being tested indicates as soon as he/she can feel the vibration and the reading on the dial at that point is recorded. The reading is

low in young normal individuals (i.e. they are very sensitive to vibration). In older individuals, the biothesiometer reading becomes progressively higher. From experience, it is known that the risk of developing a neuropathic ulcer is much higher if a person has a biothesiometer reading greater than 30-40 volts.

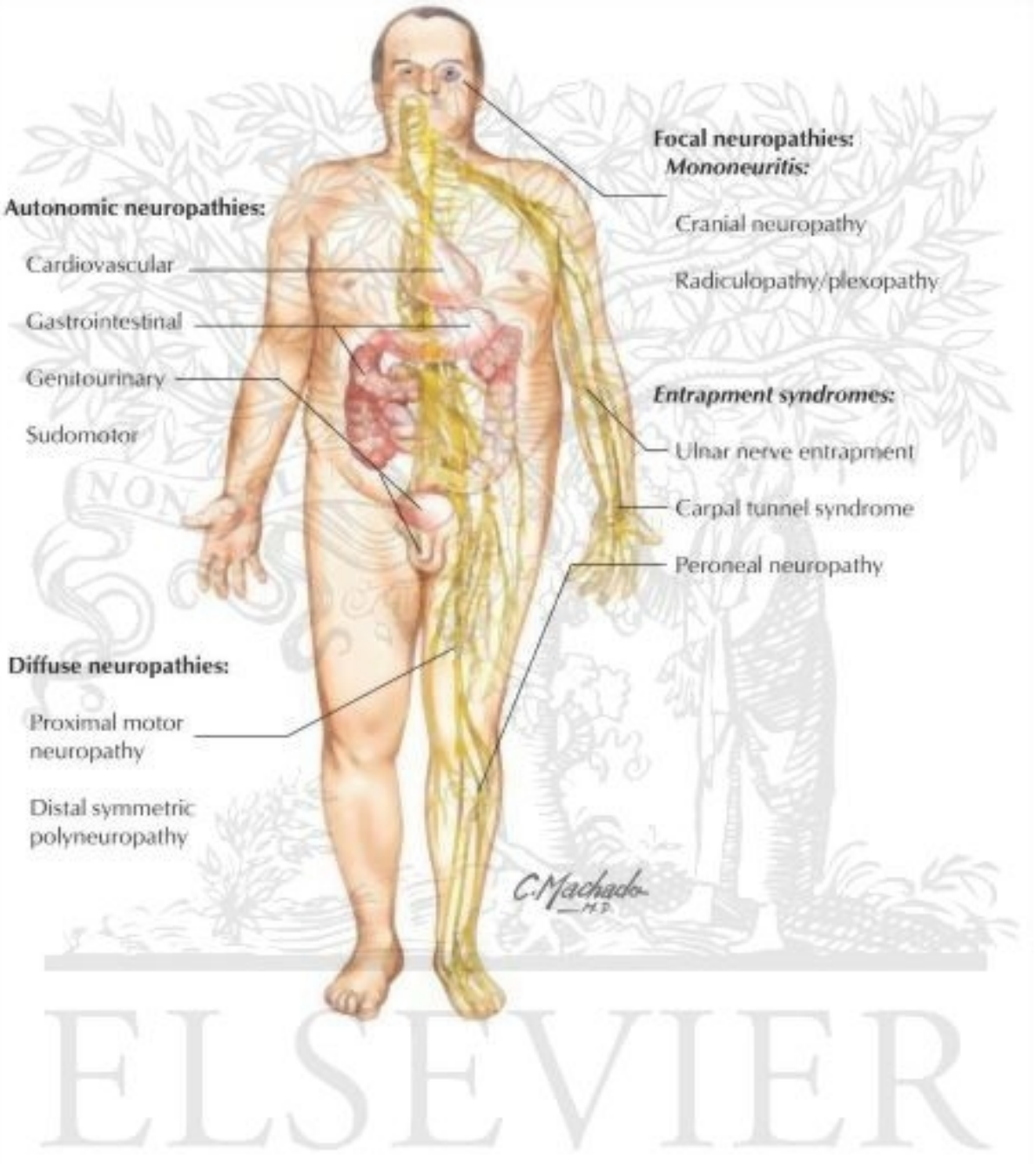
MONOFILAMENT TEST:

The 'Touch-Test' Sensory Evaluation (Semmens-Weinstein Monofilaments) application guidelines:

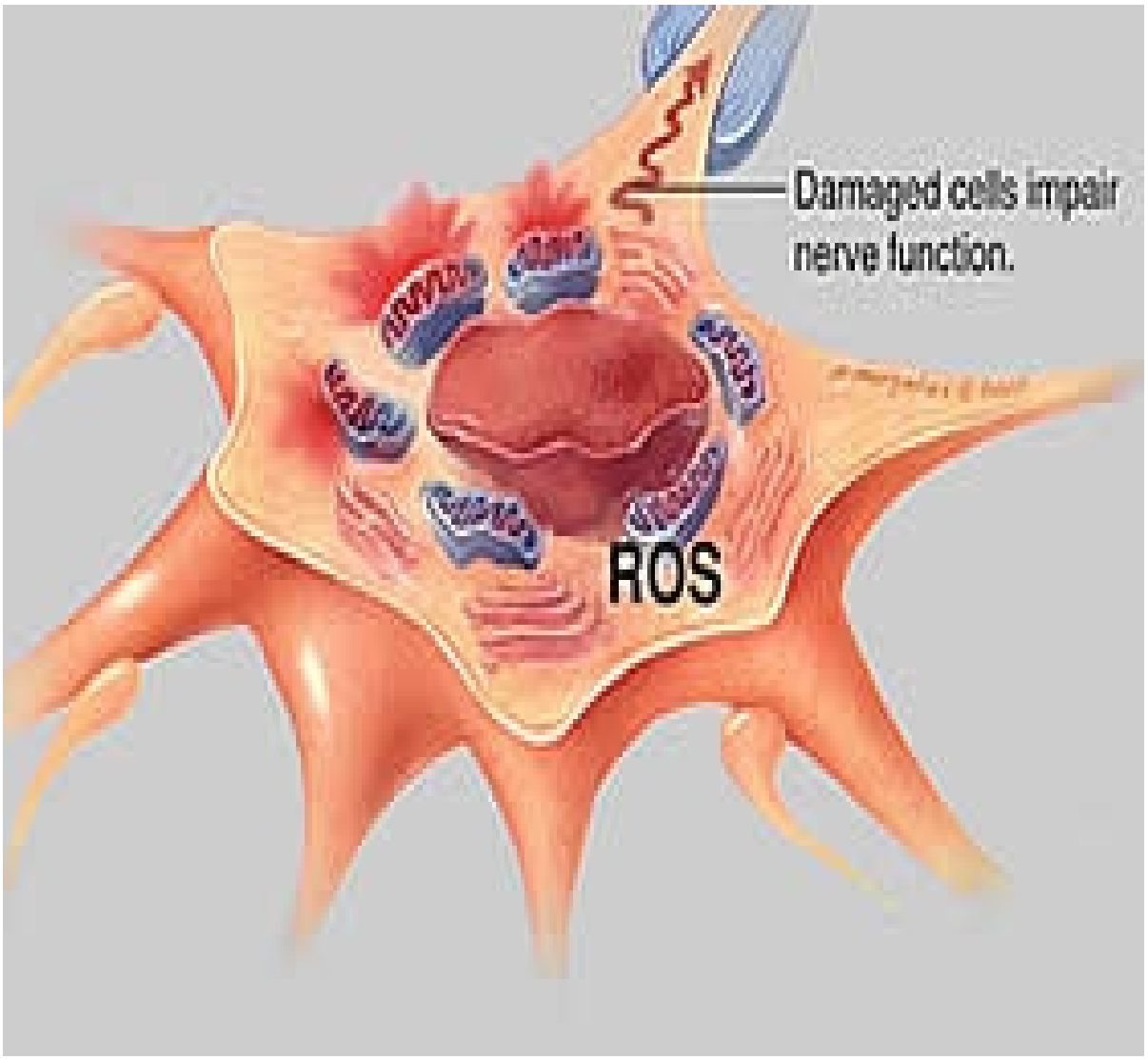
- Occlude the patient's vision by using a shield or by having the patient look away or close his or her eyes.
- Instruct the patient to respond when a stimulus is felt by saying 'touch' or 'yes'.
- Prepare to administer the stimulus to the foot (dorsal or plantar surface).
- Press the filament of the Touch
- Test at a 90 degree angle against the skin until it bows. Hold in place for approximately 1.5 seconds and then remove.

To assure the validity of the sensory test findings:

- The patient must not be able to view the administration of the stimuli so that false indications are avoided.
- The nylon filament must be applied at a 90 degree angle against the skin until it bows for approximately 1.5 second before removing.
- If the patient does not feel the filament, then protective pain sensation has been lost.



NEURONS



TRIAL MEDICINE

¸Õí¸¡Ä -ACACIA CATECHU

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KINGDOM : PLANTALE

ORDER : FABALES.

FAMILY : FABACEAE.

GENUS : ACACIA.

SPECIES :CATECHU

Synonyms:

Acacia catechu

Acacia catechuoides

Acacia sundra

Acacia walichiana

Mimosa catechu

Names in various languages:

- ✓ English: black catechu
- ✓ Malay: kachu
- ✓ Latin:catechu
- ✓ Telugu:Chandra
- ✓ Malayalam: karingali
- ✓ Sanskrit: khandira
- ✓ Hindi: katha
- ✓ Duk: kher

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þ¾É¡ø ¾Õõ §¿¡ö¸û

- ¦ÀÕ§¿¡ö-leprosy
- «Æø Ì ýÁõ-acid peptic disease
- ¦ÀÕÅÂ¢Ú- ascitis
- ÅÂ¢üÚô ÒØ §¿¡ö- helmenthic diseases
- **Ì Õ¾ì Ì ¨ÈÅ¡ø Åó¾ ¾Á÷-neuritis due to anaemia**
- **¾Á÷Å¡¾õ- neuritis due to any cause**
- **¿¡¢Æ¢× -diabetes mellitus**

CHEMICAL COMPOSITIONS:

- ✓ Catechin
- ✓ Epicatechin — - anti oxidents
- ✓ Catechunic acid — immune madulators
- ✓ Atzelchin — anti- viral
- ✓ Catechinic tetramer — anti inflammatory action
- ✓ Dicatechin
- ✓ Gallochin
- ✓ Gossypetin- anti inflammatory action
- ✓ Epiafzelechin

ACTIONS:

- ❖ ANTI DIABETIC.
- ❖ ASTRINGENT
- ❖ ANTI DYSENTERIC.
- ❖ ANTHELMENTIC.
- ❖ CURES HAVIENESS.
- ❖ ANTI CHOLESTEROL.

- ❖ ANTI ULCER
- ❖ ANTI INFLAMMATORY
- ❖ APHRODISIAC
- ❖ IMMUNO MODULATOR

IMPORTANT CHEMICAL COMPOUND:

“EPICATECHIN”- ACTION

- ✓ PROMOTE REGENARATION OF BETA CELLS OF LANGERHANS.
- ✓ CAPILLARY STABILIZING AGENT.
- ✓ VASO PROTECTIVE AGENT.
- ✓ IMPROVES BLOOD FLOW IN VESSELS.
- ✓ NATURAL SOURCE OF VITAMIN B12

“FLAVONOIDES”- PAIN KILLERS

- ✓ INHIBITS THE COX-2
- ✓ AN ANTI-OXIDENT

KARUNGALI VER

METERIALS

&

METHODS

METERIALS & METHOD
PROTOCOL

STUDY DESIGN:

The open clinical trial on VATHA KARSANAM was conducted at the OPD section of post graduates department of Pothu Maruthuvam , Government Siddha Medical College, Arignar Anna Hospital, Chennai – 600 106 during the period of 2011-2013

SAMPLE SIZE:

During this dissertation work on VATHA KARSANAM (Diabetic neuropathy) totally 40 patient of both sexes in the age group of above 40 are taken **.**

SELECTION CRITERIA:

- Only diabetic patients more than 5 year
- Both sexes
- Obesity
- Age above 40
- Willing to give specimen of blood for investigation when required.
- Willing to admitted in the hospital for one month or willing to attend the OPD once in 7 days

EXCLUSION CRITERIA:

- Idiopathic
- Hereditary
- Amyloidosis
- Anaemia
- Lyme disease
- HIV
- Metal poisioning
- Gullian – Barrie syndrome

- Fabry's disease
- Tangier's disease
- Pregnant & lactating mothers

WITHDRAWAL CRITERIA:

Didn't take the medicines regularly

Didn't turn for follow up once in a week

Development of any infectious disease or side effects during their trial period

EVALUATION OF CLINICAL PARAMETERS:

Patients are clinically evaluated by following parameters.

Siddha system of clinical diagnosis

Poriyal Therthal – Mei, Vai, Kann, Mooku, Sevi

Pulanal Therthal – Unarthal, Suvaithal, Parthal, Mugarthal, Kettal.

Venaathal

Mukkutra Nilaigal – Vali, Azhal, Iyam

Ezhu Udal Kattugal – Saaram, Senneer, Oon Kozhuppu, Enbu, Moolai, Sukkilam.

Envagai Thervu – Naa, Niram, Mozhi, Vizhi, Nadi, Sparisam, Malam, Moothiram.

Case Sheet Proforma:

Patients will be treated with clinical signs and symptoms of Madhu megam.

Complaints and Duration

History of Past illness

Personal History

Personal Habits

Family History

- Systemic Examination

- ❖ Laboratory Investigation
- ❖ Prognosis of the disease and management.

INVESTIGATION:

The next step in Research oriented programme is investigation, to confirm the diagnosis predicted. The investigations are carried out promptly and regularly before and after treatment.

All patients are subjected to routine clinical investigation which include

- ❖ Urine sugar-fasting and post – prandial
- ❖ Albumin and deposits in urine
- ❖ Total count, differential count, Erythrocyte sedimentation rate, Haemoglobin and urea, cholesterol in blood.
- ❖ Blood Sugar, fasting and post prandial ,other investigations like glucose tolerance test, glycosylated Haemoglobin (HbA1c)
- ❖ Lipid profile
- ❖ Biothesimetry
- ❖ Monofilament test with 10microgram filament
- ❖ Nerve conduction study (for affordable patients only).

TRIAL DRUG:

KARUNGALI VER KUDINEER

INGREDIENTS:

KARUNGALI VER- ROOT OF ACACIA CATECHU

DOSAGE:

30 ml BD (20 mins before food)

TRIAL PERIOD:

40 days

KARUNGALI VER KUDINEER CHOORANAM

RESULTS

&

OBSERVATION

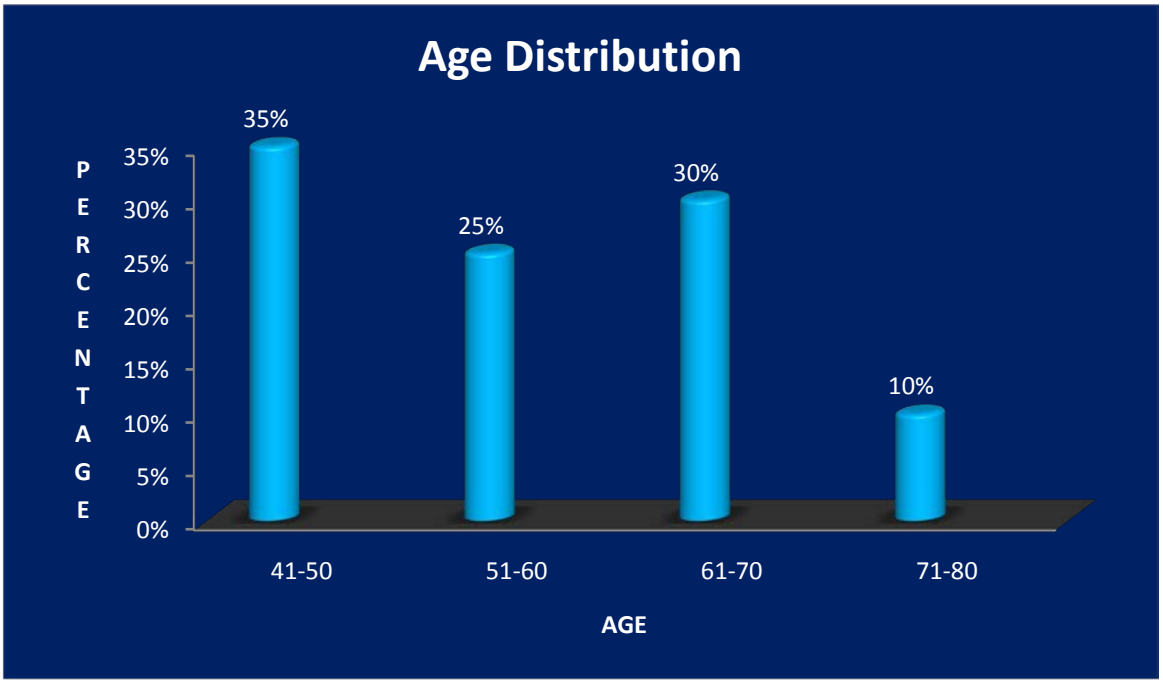
RESULTS AND OBSERVATIONS

- The factors considered for the purpose of the study comprised of the following:

- Age Distribution
- Gender distribution
- Thinai
- Paruvakaalam
- Occupational status
- Socio economic Status
- Food habits
- Personal habits
- Symptoms
- Classifications of results according to Vali, Azhal & Iyyam
- Udal kattugal
- Enn vagai thervu
- Naadi
- Classification on the basis of Neikuri
- Clinical progress
- Urine sugar(F)
- Urine sugar(pp)
- Blood sugar(F)
- Blood sugar(pp)
- HbA1C
- Monofilament test
- Results after treatment.

AGE DISTRIBUTION

SL.NO	AGE	NO. OF PATIENTS /20	PERCENTAGE
1.	41-50	14	35%
2.	51-60	10	25%
3.	61-70	12	30%
4.	71-80	4	10%

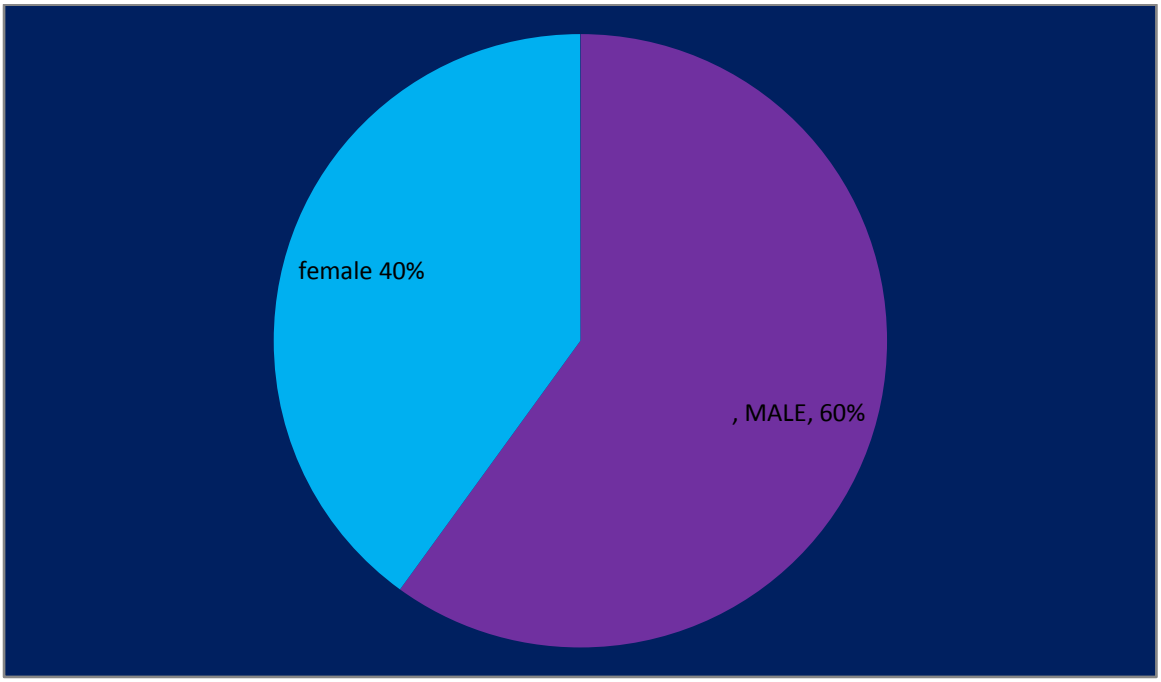


Inference:

According to the above mentioned data 30% of patients were in age groups 61-70 years, 10% of patients were in age group 71-80 year, 25% of patients were in age group 51-60 years and 35% of patients were in age group 41-50 years.

GENDER WISE DISTRIBUTION:

GENDER	NO.OF PATIENTS	PERCENTAGE
MALE	24	60%
FEMALE	16	40%



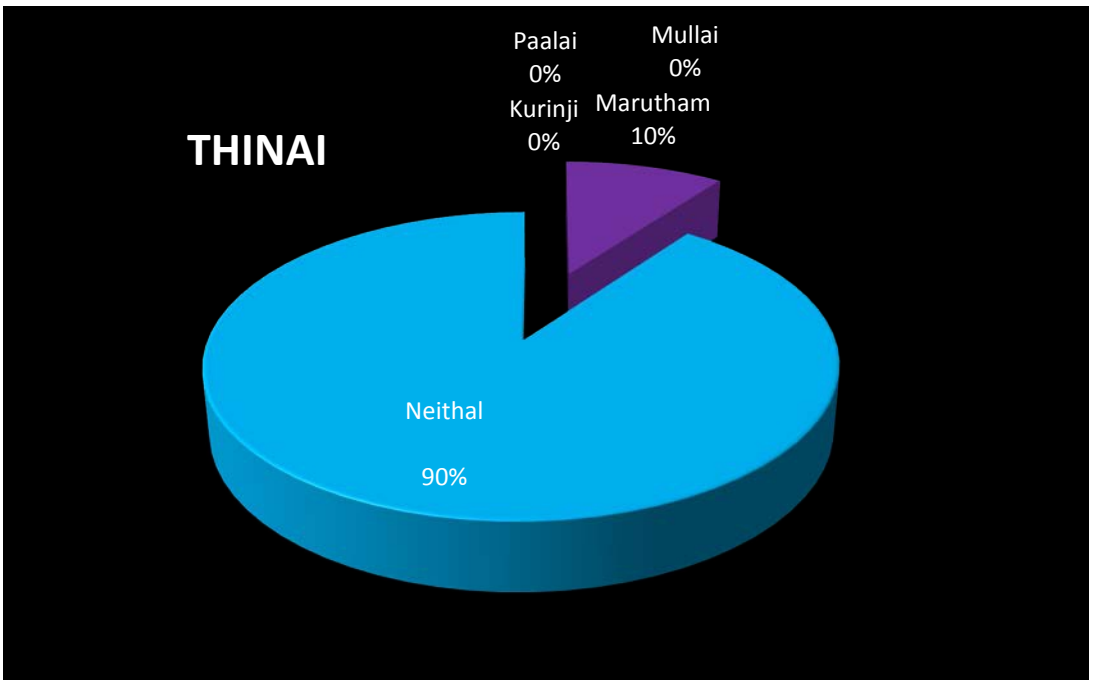
INTERFERENS:

60% affected were men

40% affected were women

THINAI:

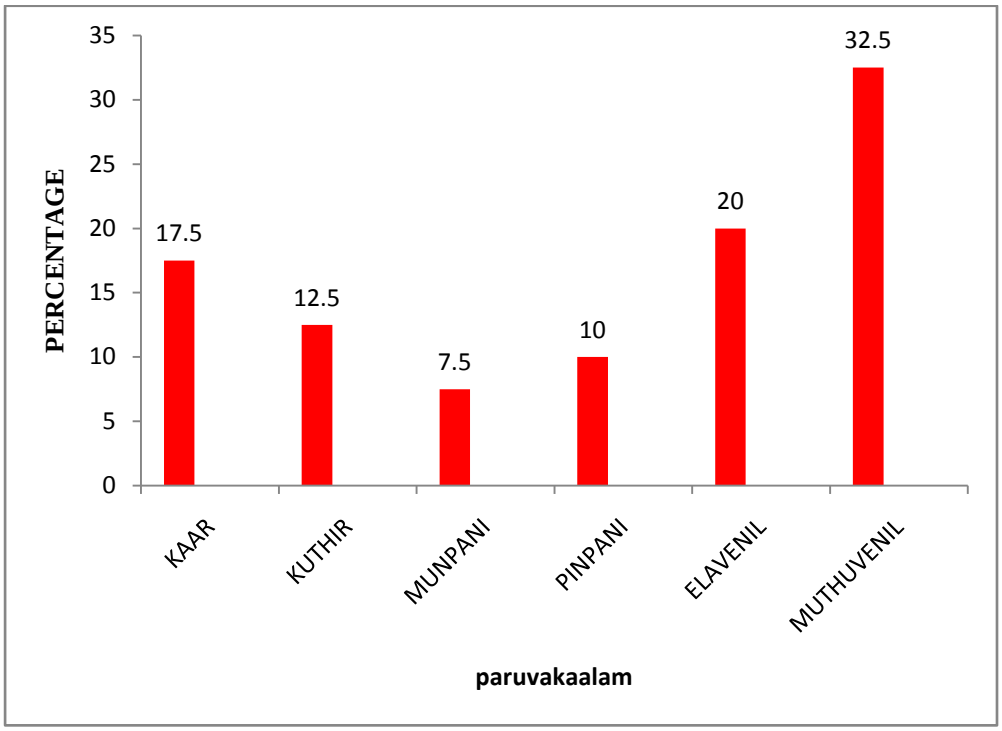
SL.NO	THINAI	NO. OF PATIENTS /20	PERCENTAGE
1.	Kurinji	0	0%
2.	Mullai	0	0%
3.	Marutham	4	10%
4.	Neithal	36	90%
5.	Paalai	0	0%



Inference:
From the above data 90% of patient from Neithal and10% of cases from marutham.

PARUVAKAALAM:

SL.NO.	PARUVAKAALAM	NO. OF PATIENTS /20	PERCENTAGE
1.	Kaar kaalam (Aavani & purattasi) Aug 16 to Oct15	7	17.5%
2.	Koothir kaalam (Iypasi &karthigai) Oct 16 to Dec15	5	12.5%
3.	Munpani kaalam (Margazhi & Thai) Dec16 to Feb15	3	7.5%
4.	Pinpani kaalam (Masi& Panguni) Feb16 to June15	4	10%
5.	Elavenir kaalam (chithirai & vaikaasi) April16 to June15	8	20%
6.	Mudhuvenir kaalam Aani & Aadi June16 to Aug 15	13	32.5%

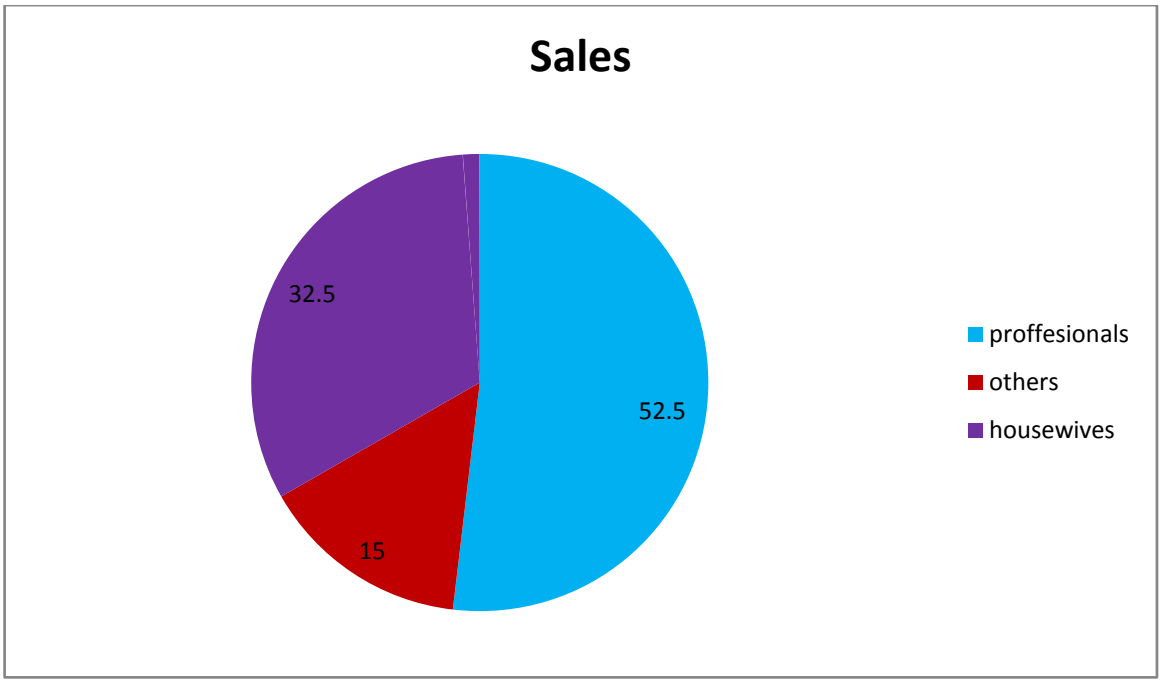


Inference:

17.5% of case came in Kaar kaalam and 12.5% of case in kuthir kaalam,7.5% of cases in munpani kaalam,10% of cases in Pinpani kaalam, 20% of cases in ilavenil kaalam, 32.5% of cases in muthuvenil kaalam.

OCCUPATIONAL STATUS

SL.NO	OCCUPATION	NO OF PATIENTS /20	PERCENTAGE
1.	Professionals	21	52.5%
2.	Others	6	15%
3.	House wives	13	32.5%



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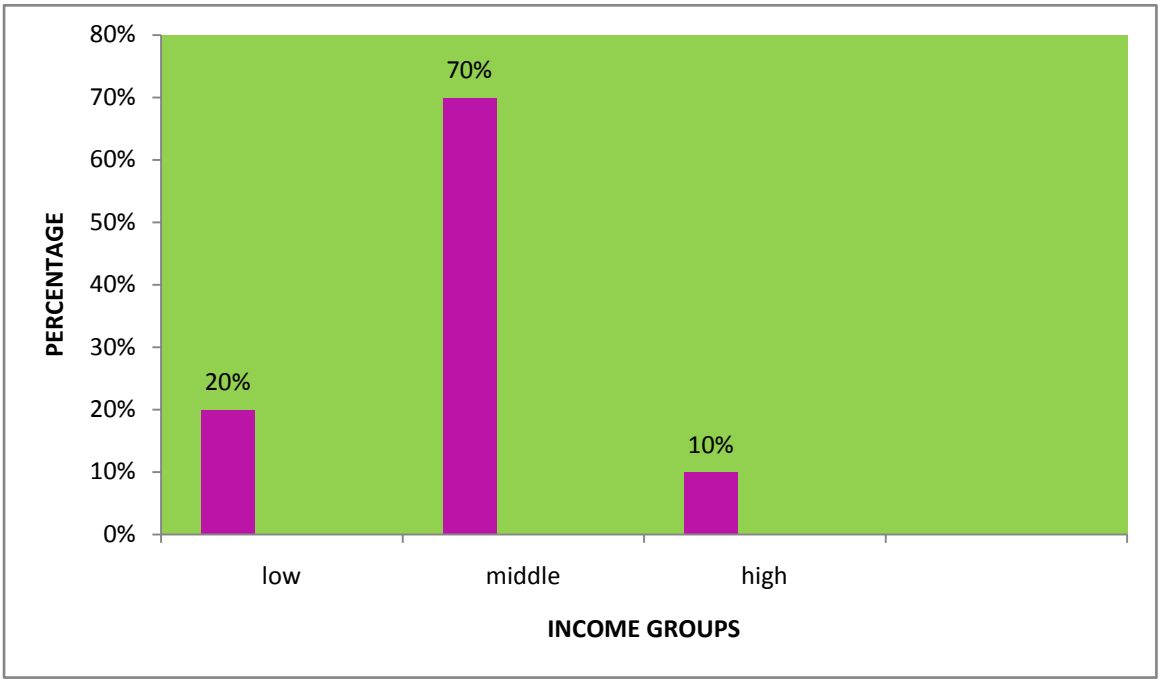
Inference:

52.5% of cases were Professionals.
15% of cases were Retired persons are coming under others.
32.5% of cases were house wives

SOCIO ECONOMIC STATUS

Sl.No.	*Socio Economic Status*	No. of Patients /40	Percentage
1.	Low income group (below 10000/month)	8	20%
2.	Middle income group (10000-20000/ month)	28	70%
3.	High income group (above 20000/month)	4	10%

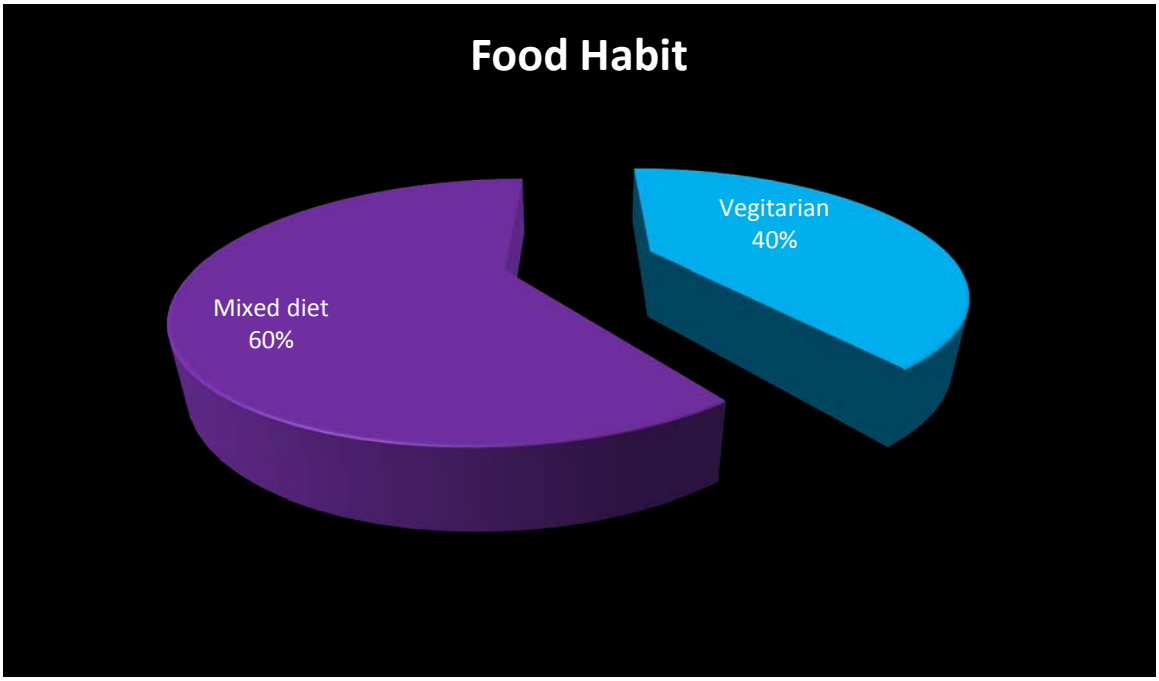


Inference:

70% of cases belong to middle income group and 20% of patients belong to lower income group. 10% of cases belong to high income group.

FOOD HABITS

SL.NO.	FOOD HABIT	NO. OF PATIENT / 20	PERCENTAGE
1.	Vegetarian	16	40%
2.	Mixed diet(including non-veg)	24	60%

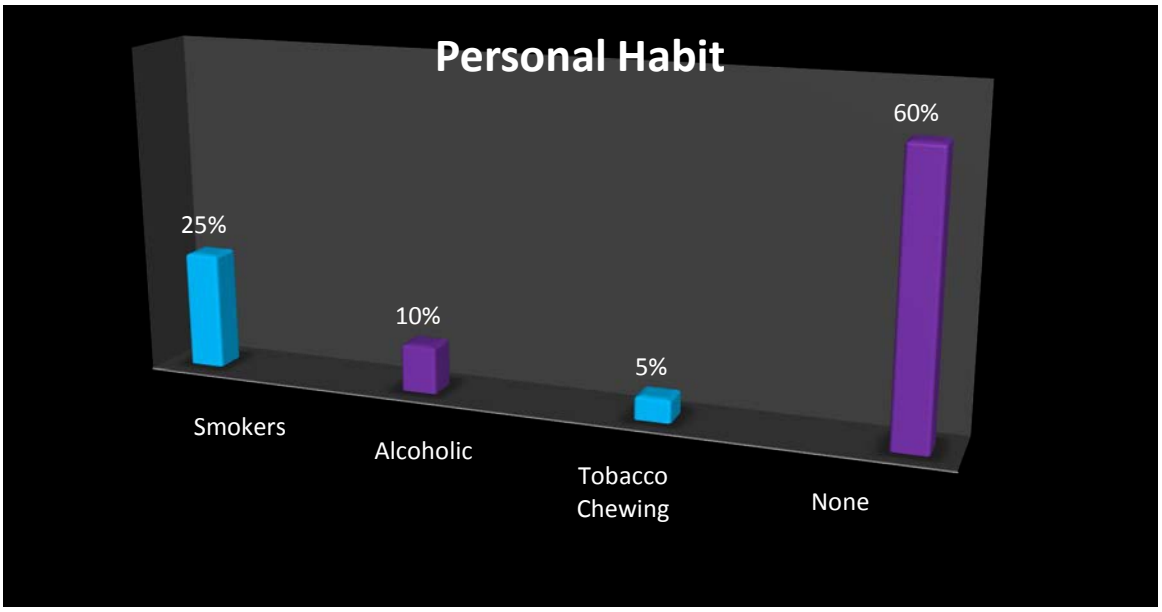


Inference:

60% of cases were mixed diet. With non veg diet
40% of cases were Vegetarian.

PERSONAL HABITS

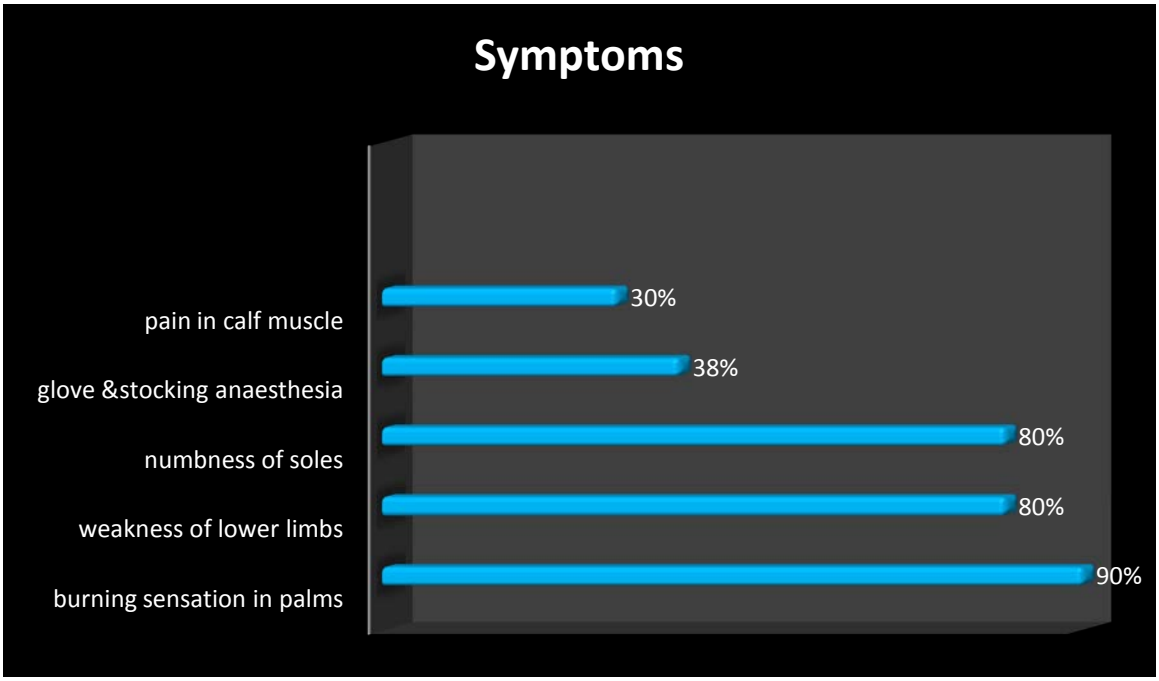
SL.NO.	PERSONAL HABIT	NO. OF PATIENTS / 40	PERCENTAGE
1.	Smoker	10	25%
2.	Alcoholic	4	10%
3.	Tobacco chewing	2	5%
4.	Others	24	60%



Inference:
60% of patients had no bad habits, 25% of cases were smoker and .10% of cases were alcoholic, 5% of cases were Tobacco Chewing.

SYMPTOMS

SL.NO	SYMPTOMS	NO. OF PATIENTS/ 40	PERCENTAGE
1.	Burning sensation in palms	36	90%
2.	Pain in calf muscles	12	30%
3.	Numbness of soles	32	80%
4.	Glove and stocking type of anaesthesia	15	38%
5.	Weakness of lower limbs	32	80%

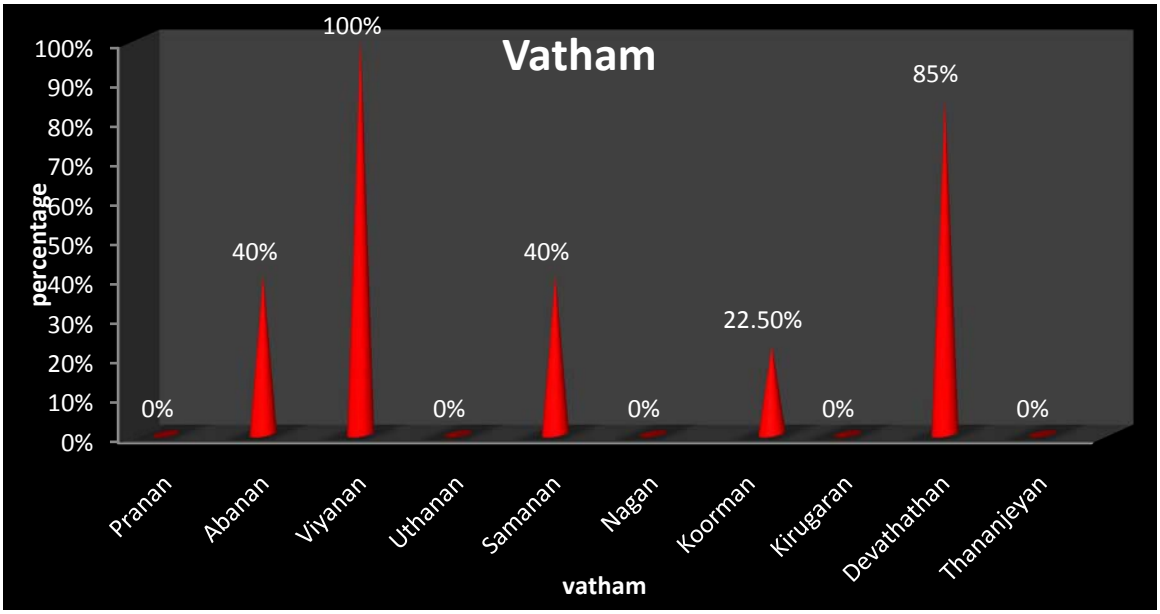


Inference:

90% of cases had burning sensation in palms 80% of cases had numbness over the soles& palms,75% 0f cases had weakness of lower limbs, 38% of cases had glove & stocking type of anesthesia 30% of cases had pain in calf muscles.

VATHAM

SL.NO.	VATHAM	NO. OF PATIENTS / 40	PERCENTAGE
1.	Pranan	0	0%
2.	Abanan	16	40%
3.	Viyanan	40	100%
4.	Uthanan	0	0%
5.	Samanan	16	40%
6.	Nagan	0	0%
7.	Koorman	9	22.5%
8.	Kirugaran	0	0%
9.	Devathathan	17	85%
10.	Thananjeyan	0	0%

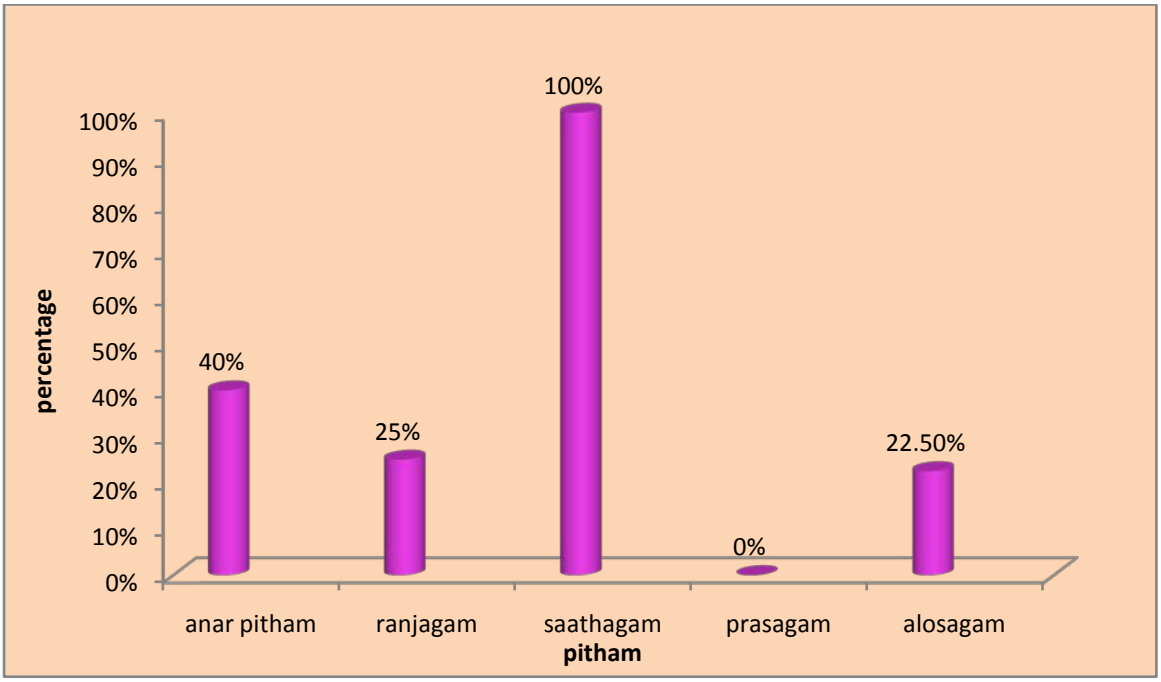


Inference:

Viyanan was affected in 100% of patients and Devathathan was affected in 85% of patient. Abanan and samanan were affected in 40% of cases. koorman affected in 22.5% of cases.

PITHAM

SL.NO.	PITHAM	NO.OF PATIENT /20	PERCENTAGE
1.	Anar pitham	16	40%
2.	Ranjagam	10	25%
3.	Saathagam	40	100%
4.	Prasagam	0	0%
5.	Alosagam	9	22.5%

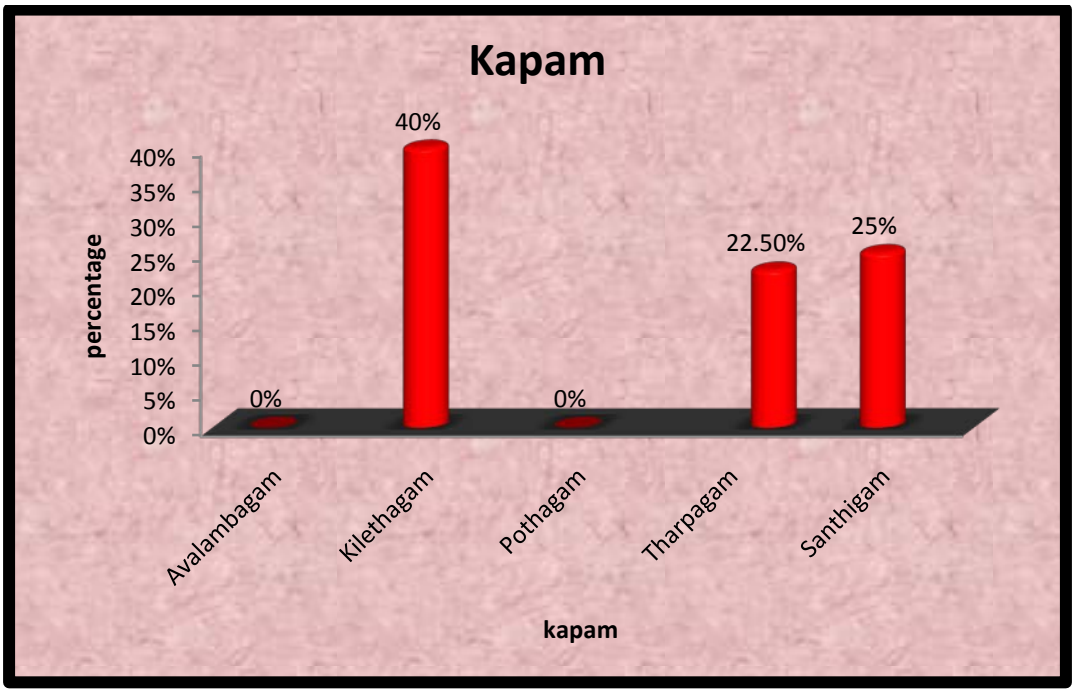


Inference:

Anarpitham was affected in 40% of patients , sathagam was affectedin 100% of patients ,alosagam was affected in 22.5% of cases. Ranjagam was affected in 25% of cases.

KAPAM

SL.NO.	KAPAM	NO. OF PATEINTS /40	PERCENTAGE
1.	Avalambagam	0	0%
2.	Kilethagam	16	40%
3.	Pothagam	0	0%
4.	Tharpagam	9	22.5%
5.	Santhigam	10	25%

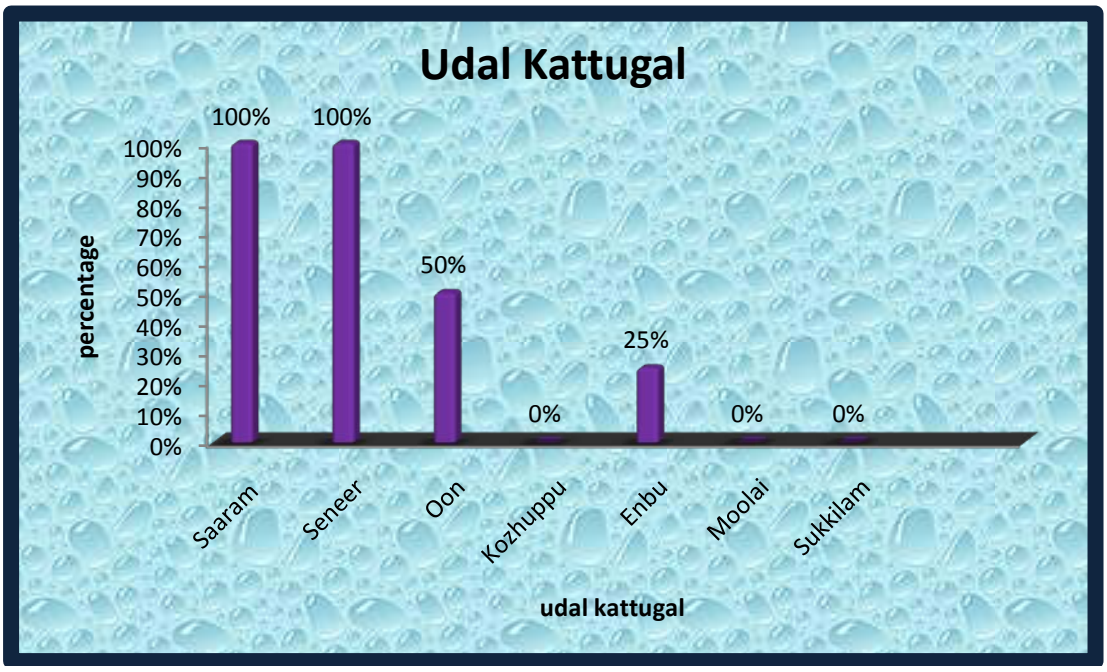


Inference:

Kilethagam was affected in 40% of patients and Santhigam in 25% of patients. tharpagam was affected in 22.5% of case

UDAL KATTUGAL

SL.NO.	UDAL KATTUGAL	NO. OF PATIENT / 20	PERCENTAGE
1.	Saaram	40	100%
2.	Seneer	40	100%
3.	Oon	20	50%
4.	Kozhuppu	0	0%
5.	Enbu	10	25%
6.	Moolai	0	0%
7.	Sukkilam	0	0%

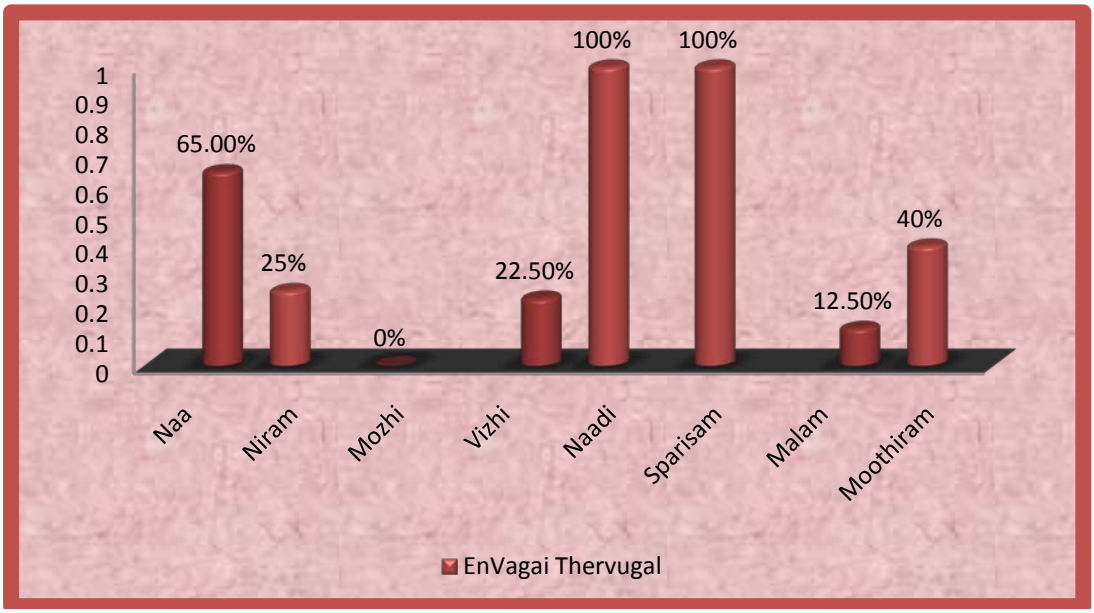


Inference:

Saram affected 100%
Seneer affected 100%
Enbu aaffected 25% Oon affected 50%

ENVAGAI THERVUGAL

SL.NO.	ENVAGAI THERVUGAL	NO. OF PATIENT / 20	PERCENTAGE
1.	Naa	26	65%
2.	Niram	10	25%
3.	Mozhi	0	0%
4.	Vizhi	9	22.5%
5.	Naadi	40	100%
6.	Sparisam	40	100%
7.	Malam	5	12.5%
8.	Moothiram	16	40%

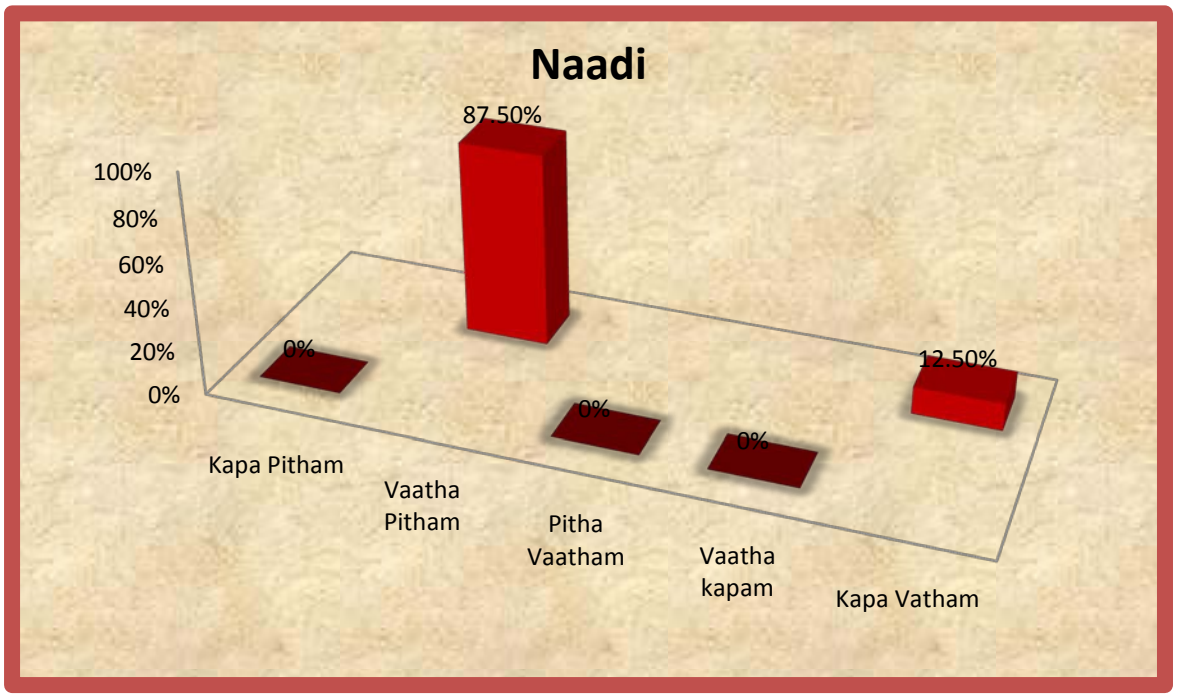


Inference:

Naadi was affected in 100% of patients and 100% of patients sparisam was affected

NAADI

SL.NO.	NAADI	NO. OF PATIENT / 20	PERCENTAGE
1.	Kappa Pitham	0	0%
2.	Vaatha Pitham	35	87.5%
3.	Pitha Vaatham	0	0%
4.	Vaatha Kapam	0	0%
5.	Kapa Vaatham	5	12.5%

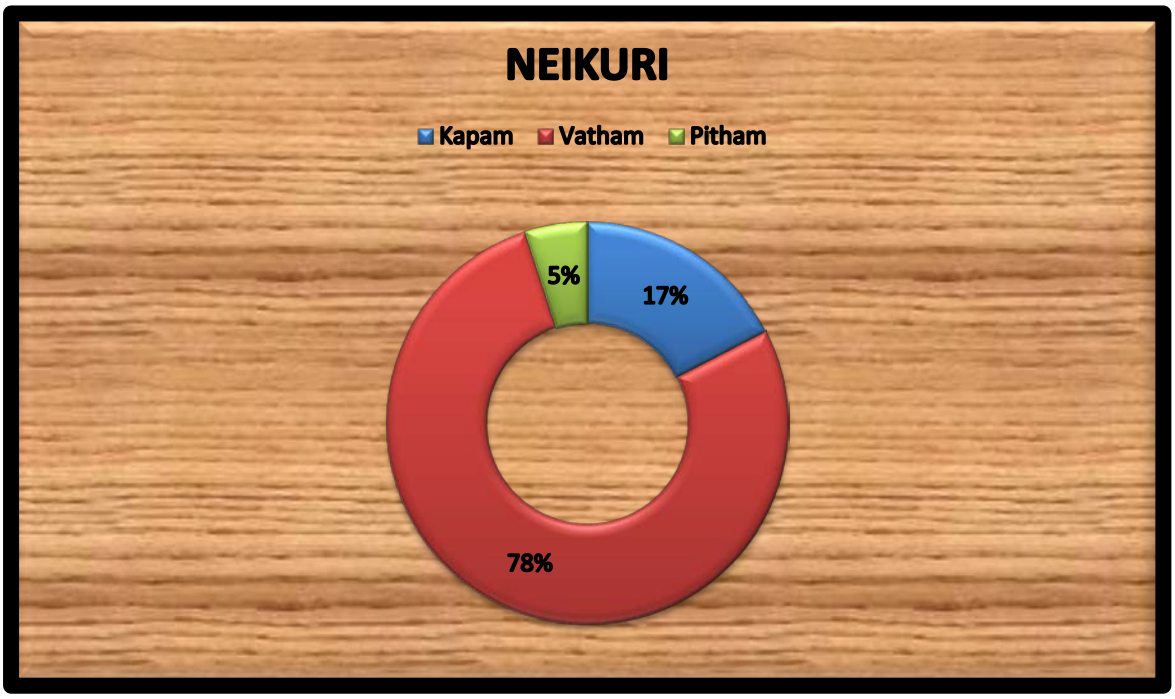


Inference:

87.5% of patient's vatha pitham naadi was felt and 12.5% of cases kabha vatha naadi was felt.

NEIKURI

SL.NO.	NEIKURI	NO. OF PATIENT / 20	PERCENTAGE
1.	Vatham (Spreads like Snake)	31	77.5%
2.	Pitham (Spreads like Ring)	2	5%
3.	Kapam (Stands like Pearl)	7	17.5%

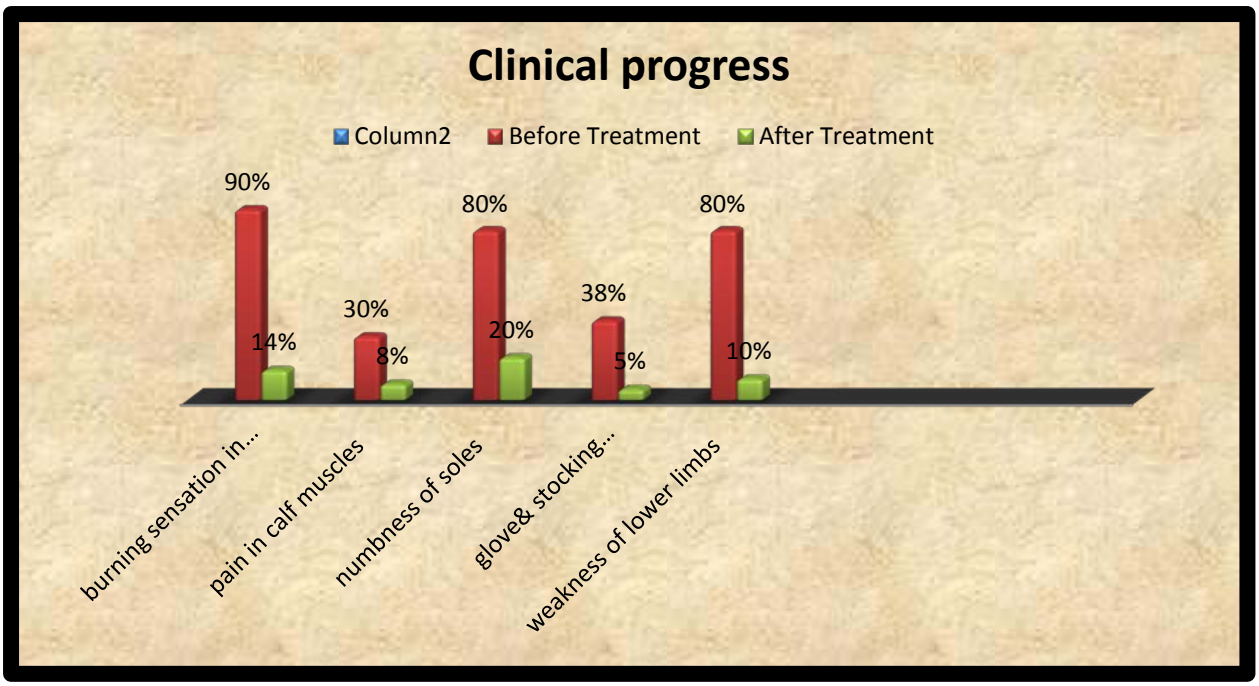


Inference:

77.5% of cases show Vatha neikuri, 17.5% shows Kapha neikuri and 05% shows Azhal neikuri.

CLINICAL PROGRESS

SL.NO	SYMPTOMS	NO. OF PATIENTS/ 40		PERCENTAGE	
		Before Treatment	After Treatment	Before Treatment	After Treatment
1.	Burning sensation in palms	36	6	90%	14%
2.	Pain in calf muscles	12	3	30%	7.5%
3.	Numbness of soles	32	8	80%	20%
4.	Glove and stocking type of anaesthesia	15	2	37.5%	5%
5.	Weakness of lower limbs	32	4	80%	10%

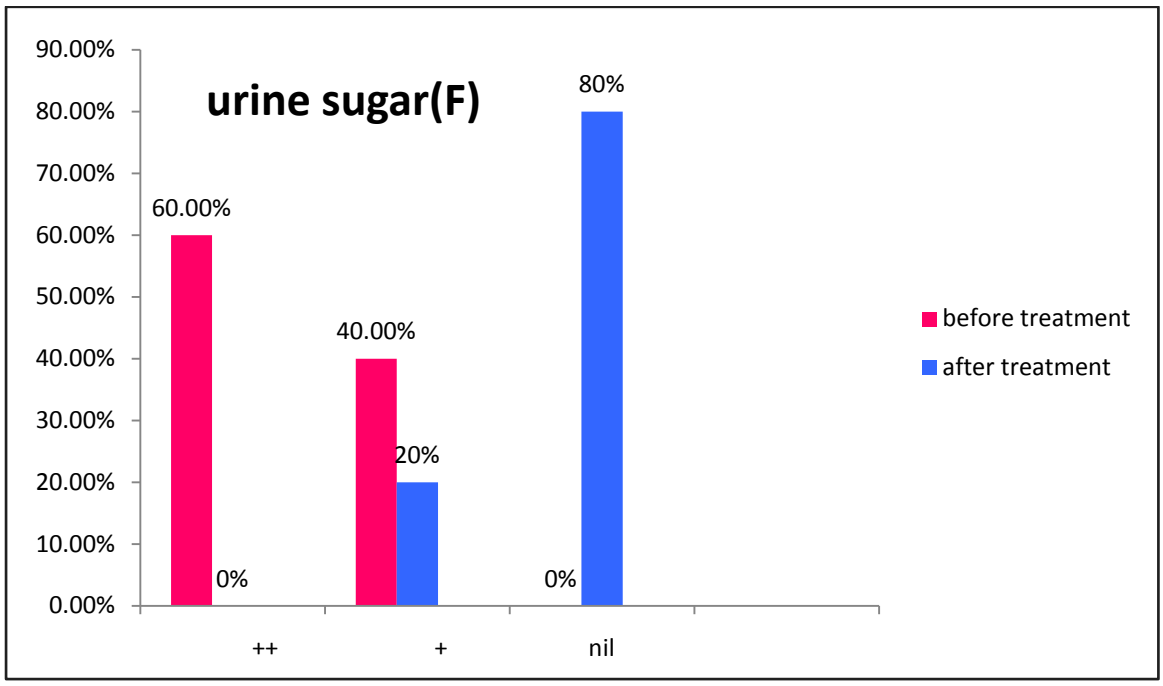


Inference

burning sensation in palms has reduced from 90% to 14%, numbness over the soles& palms had reduced from 80% to 20%, weakness of lower limb had reduced from 80% to 10%, glove & stocking type of anesthesia has reduced from 37.5% to 5%, pain in calf muscles has reduced from 30% to 8%.

URINE SUGAR LEVELS (FASTING)

URINE SUGAR (FASTING)	Before treatment	percentage	After treatment	percentage
++	24	60%	0	0%
+	16	40%	8	20%
nil	0		32	80%

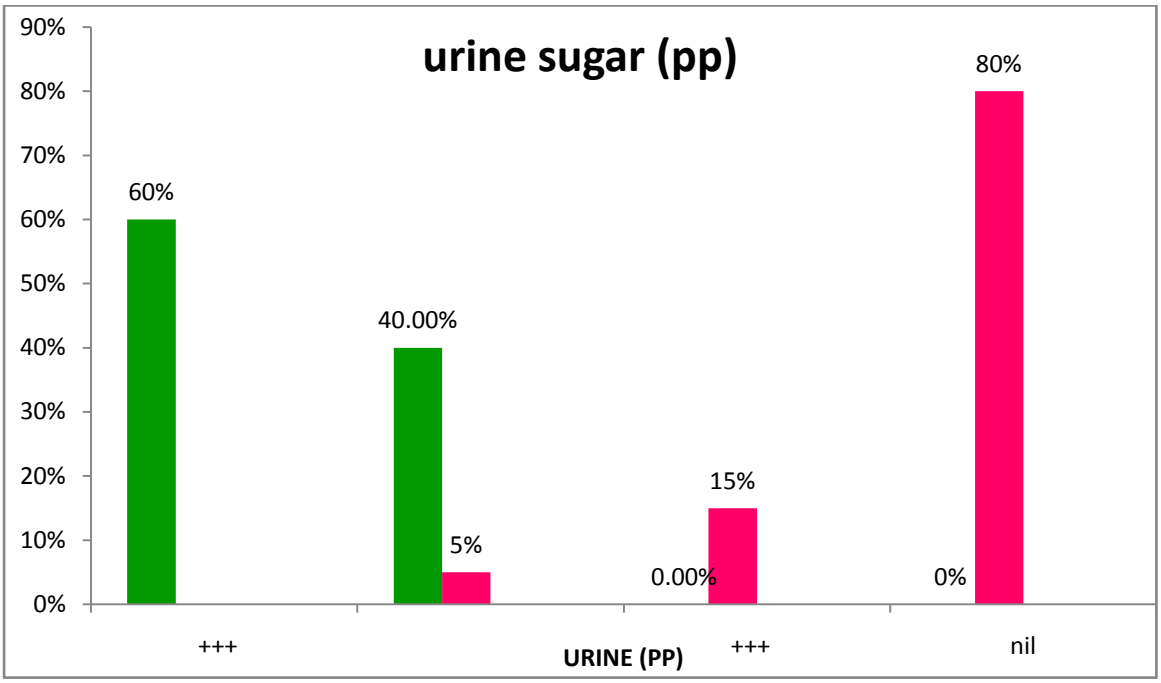


Inference:

Urine (F) becomes nil in 80% 0f cases.

Urine sugar(pp)

Urine sugar (pp)	Before treatment	percentage	After treatment	Percentage
+++	24	60%	0	0%
++	16	40%	2	5%
+	0	0%	6	15%
nil	0	0%	32	80%

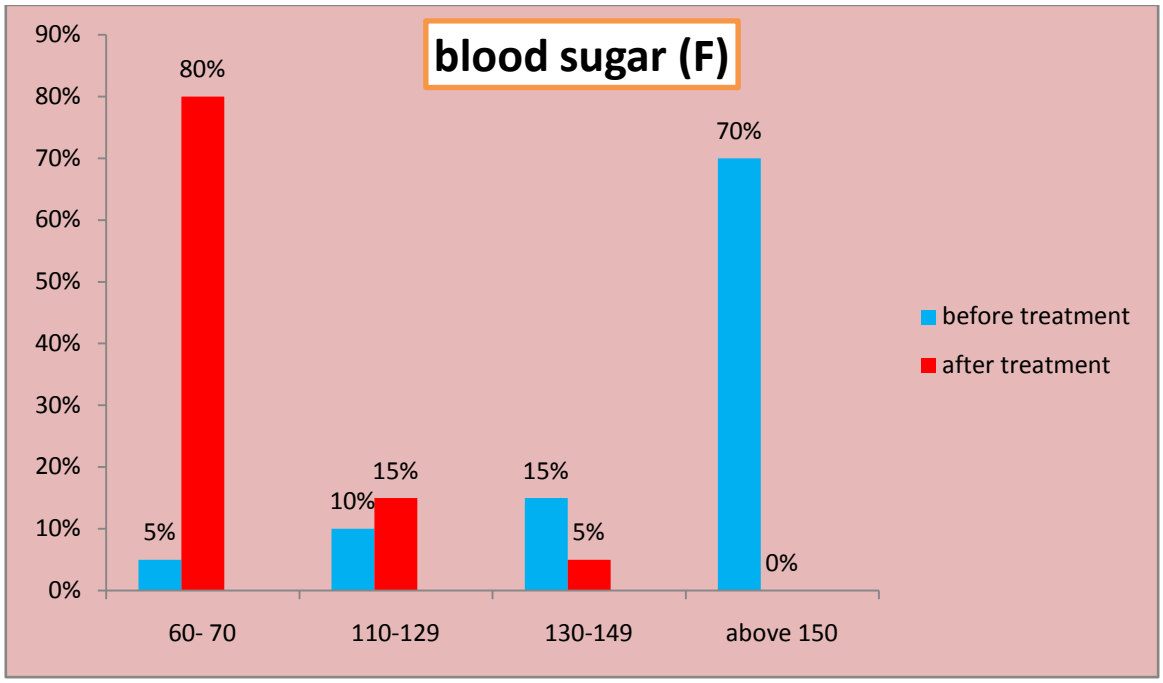


Inference:

In 80% of cases the urine sugar (pp) becomes nil.

Blood sugar (fasting)

Blood sugar(fasting)mg/dl	Before treatment	percentage	After treatment	percentage
60-109	2	5%	32	80%
110-129	4	10%	6	15%
130-149	6	15%	2	5%
Above 150	28	70%	0	0%

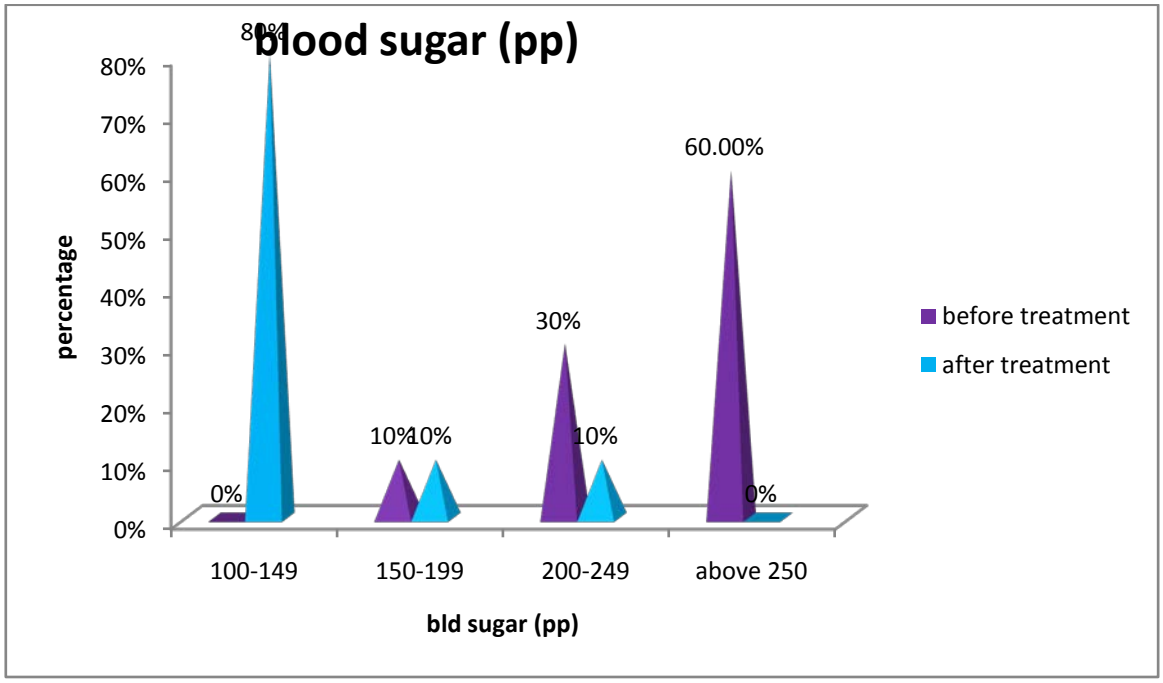


Inference:

After treatment the fasting blood sugar level fall within 60-70mgs/dl in 80% of cases

Blood sugar (post prondial)

Blood sugar (pp) mgs/dl	Before treatment	percentage	After treatment	percentage
100-149	0	0%	32	80%
150-199	4	10%	4	10%
200-249	12	30%	4	10%
Above 250	24	60%	0	0%

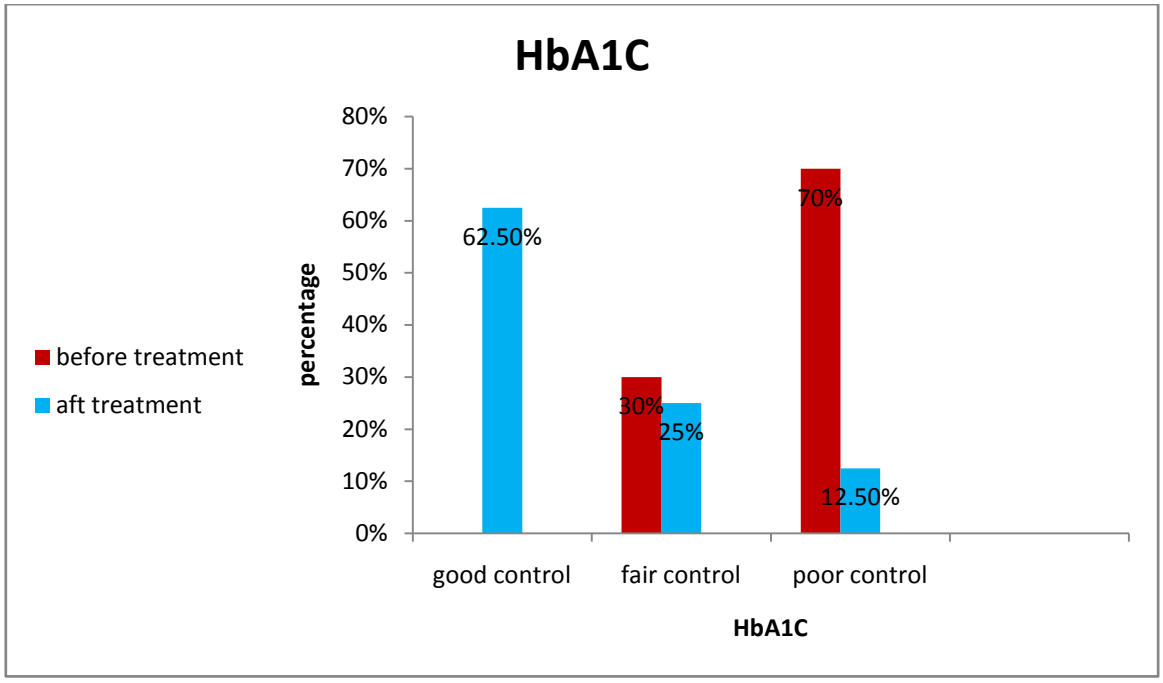


Inference :

The pp blood sugar levels falls between 100-149mgs/dl in 80% of cases

HbA1C REPORT

HbA1C	Before treatment	percentage	After treatment	percentage
Good control 5.7-7%	0	0%	25	62.5%
Fair control 7-8%	12	30%	10	25%
Poor control Above 8%	28	70%	5	12.5%

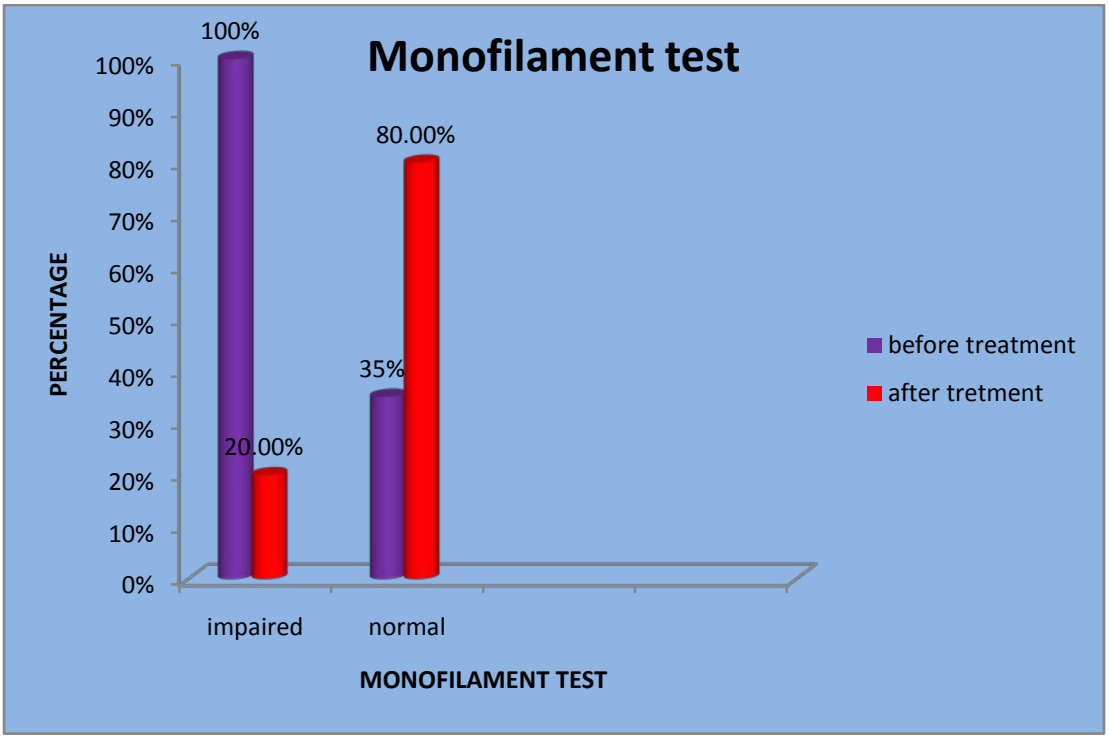


Inference:

HbA1C reports shows the improvement of good control from 0% to 62.5%

MONOFILAMENT TEST:

Monofilament test with 10mg filament	Before treatment	percentage	After treatment	percentage
Impaired	40	100%	8	20%
Normal	0	0%	32	80.%

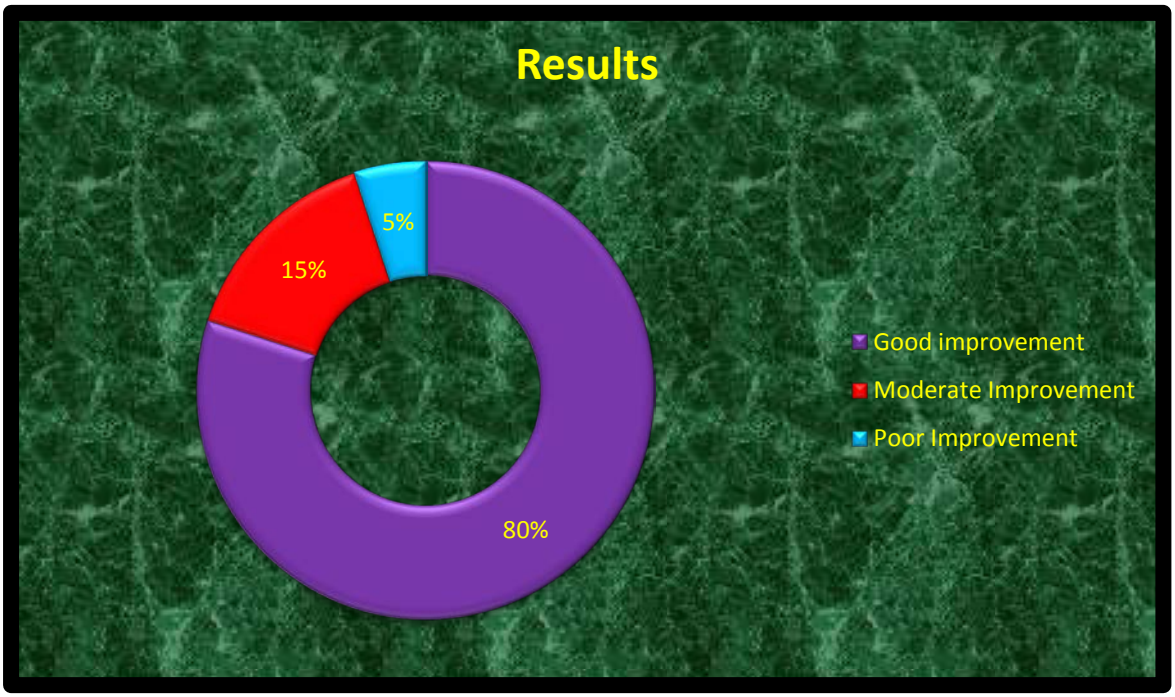


Inference:

80% of cases get normal in monofilament test after treatment.

PROGNOSIS

SL.NO.	Results	NO. OF PATIENT / 40	PERCENTAGE
1.	Good improvement	32	80%
2.	Moderate Improvement	6	15%
3.	Poor Improvement	2	5%



Inference:

Based on blood sugar level and mono filament test 80% of Patients show good improvement, 15% of shows moderate improvement and 5% of cases shows poor improvement

DISCUSSION

DISCUSSION

The sweeping change in the life style and the food habits of people in the modern world has absolutely played a significant role in the health aspect of humanity.VATHA KARSANAM is the disorders of peripheral nerves either sensory motor or mixed symmetrical and affecting distal parts of the limbs more than proximal.About26% of world diabetic populations having neuropathy. In this study, the various factors were taken into consideration with the small group of forty patients. The aspects that were looked into for study are discussed as follows:

AGE WISE ANALYSIS:

The study shows that the vathakarsanam (dm neuropathy)is common among the age group of 40-80. In 41-50years- 35% cases, 51-60 years- 25%, 61-70years -30% of cases & 71-80years- 10%. Being the complication of diabetes it occurs in above all the age depending upon the control of blood sugar.

GENDER WISE ANALYSIS:

It shows that men are mostly in poor control of diabetes than female. i.e. male-60%, female-40%. But usually occurs in both sexes.

THINAI(LAND):

According to this study 90% of cases reported in patients who live in Neithal. Remaining 10% cases reported from marutham . Moreover neithal land makes people obese which cause the disease (Siddha Maruthuvanga Churukkam – P - 256).

PARUVA KAALAM (SEASONAL INCIDENCE):

The seasonal changes does not have any impact on this study. But most of the cases reported in muthuvenil kallam-32.5%, kaarkalam-17.5%,munpanikaalam-7.5%,kuthir kaalam-12.5%, pinpanikaalam-10%, elavenil kaalam-20%.

OCCUPATIONAL ANALYSIS:

Due to sedentary life and irregular food habits working people (52.5%) having high rate of incidents. Next housewives (32.5%) having higher incidents due to lack of physical work. Retired people having 15% of incidences..

SOCIO ECONOMIC STATUS:

People belonging to all groups are affected in vathakarsanam. Middle income groups were reported 70% in this study. Low income group was affected 20%,high income group cases affected 10%.

DIETARY HABITS:

Vatha karsanam is more among all groups particularly in mixed diet (including non-vegetarian diet) people. Fast food & junk food habits aggravated the symptoms of the disease due to increase of blood sugar level**,**

“உற்பவிக்கும் பால் நெய் யால் இறைச்சி கள்ளால்”

– யூகி வைத்திய சிந்தாமணி பக்கம் – 146

As per Agasthiar,

“கொழுத்த மீனிறைச்சி போதை

ஒது நீரிழிவு சேர”

– சித்த மருத்துவம் – பக்கம் 470

MUKUTTRAM CLASSIFICATION:

VALI: viyanan , devathathan, , abanan are affected.

The action of abanan is to regulate the function of excretion of urine and feaces. The derangement of the function of abana leads to polyuria and constipation. It was deranged in 40% cases.

Viyanan exists all over the body, it lives in skin and activate the 72000 Nadi's. It helps the movement of all organs and sensation. In vathakarsanam viyanan is affected and so it leads to pain all over the body, pain and pricking sensation numbness of palms & soles. It was deranged in 100% of cases.

Devathathan is responsible for general ability of body and mind it was affected in 85% of cases. Derangements of this vatham leads to weakness of lower limbs and tiredness.

Koorman was affected in 22.5% of cases. It is responsible for vision.

AZHAL:

Anar pitham is affected in 40% of cases. In general Anar pitham action is exactly between the stomach and small intestine which means the pancreatic action is mainly maintained by Anarapitham. Its derangement causes DIABETES.

Sathaga pittham is affected in 100% of patients.

Alosaga pittham is responsible for vision . It is affected in 22.5% due to cataract.

Ranjaga pitham is responsible for changes in blood. It was affected in 25% cases.

KABHAM:

Kilethagam was affected in 40% cases. The function of Kilethagam is to make the contents (food) of the stomach ready for digestive process. If the function get deranged the initial phase of metabolism gets affected.

Santhigam affected in 25% of cases. Tharpagam was affected in 22.5% due to vision distrubences.

EZHU UDAL KATTUGAL:

Vatha karsanam saram, seneer, oon,enbu are affected.

Saram, seneer were affected in 100% of patients. Oon was affected in 50% of cases. Enbu was affected in 25% of cases.

ENVAGAI THERVUGAL:

NAADI & SPARISAM were affected in 100% of patient. Naa affected in 65% of cases, moothiram affected in 40% of cases.vizhi affected in 22.5% 0f cases. Niram affected in 25%.

NEIKURI:

78% of patient vatha neikuri.17.% kabha neikuri.5% of pitha neikuri.

NAADI:

In all the patient vaatha thontha naadi is prominent.

i.e kapha vatham- 12.5%

vatha pitham-87.5%

SIGNS AND SYMPTOMS:

The main symptoms of vathakarsanam burning sensation, pain in calf muscles, numbness of soles, glove & stocking type of anaesthesia, weakness of lower limbs. All the symptoms has markedly improved clinically and their conditions was good.

Urine sugar fasting & post prandial has become normal in 80% of cases.

Blood sugar fasting & post prandial has improved 75% and 70% of the cases respectively.

Blood sugar in the range of above 250mgs/dl shows good response

HbA1C shows good control in 62.5% of cases shows the precious management and control of vathakarsanam.

Mono filament test:

This shows high percentage of improvement in 80% 0f cases.

TREATMENT: MUKKUTRA THEORY

The disease and treatment are based primarily on the derangement of UYIR THATHUKKAL which again is based on theFIVE ELEMENTS theory. Incidence of VATHA KARSANAM and treatment are also based on these primary principles of Siddha medicine.

"THUVARPPU SUVAI"-MAAN + KATTRU

In vatha karsanam vali and thee pootham are commonly affected.THUVARRPU SUVAI mainly composed of kattru that is vali. So the drug act on **OPPURAI** basis.

The fusion of Thee +Thee gives raise to pitham. If there is excess of these boothas in the body, it is excreted in the urine, dryness of mouth. The medicine chosen for the treatment of vathakarsanam has "THUVARPPU SUVVAI" which is having the property of settle down the vatham as well as the pitha kuttram. There by the drug plays an important role on vathakarsanam**.** So the trial drug was act on **ETHIRURAI** basis .

According to above theory the drug acts in the basis of **KALAPPURAI** basis.

Based on the animal study done in vels university reveals that KARUNGALI VER KUDINEER is safe for prolong use for a life time disease madhumegam (diabetes).

QUALITATIVE ANALYSIS:

- The sodium corbonate extract of the medicines were tested for acid radicals, basic radicals and miscellaneous compounds.
- The results shows karunkali ver kudineer presence of iron,chloride, zinc, and reducing sugar.

TOXICOLOGICAL STUDY:

- Acute and sub acute toxicity studies were conducted on experimental rats at vels college of pharmacy.
- The hematological parameters, liver function test, renal function test and histopathology of vital organs shows no toxicity effect for KARUNGALI VER KUDINEER.

PHARMACOLOGICAL STUDY:

KARUNGALI VER KUDINEER shows significant analgesic effect in higher doses

STATISTICAL ANALYSIS:

- In both subjective and objective parameters were statistically significant.
- The statistical analysis reveals that the drug has good potency to treat the disease vatha karsanam.

SUMMARY

SUMMARY

The clinical study on VATHA KARSANAM was carried out in Post graduate department of Maruthuvam, Government Siddha Medical College, Arignar Anna Hospital, Chennai –106 during the period of 2011-2013.

A total of 40 patients were treated in the O.P and I.P department. The clinical and pathological assessment was carried out on the basis of both Siddha and modern aspects.

All the 40 patients were treated with KARUNGALI VER KUDINEER (30 ml 20 minutes before food). The duration of the treatment was fixed as 45 days. The responses were assessed 7 days once for all the patients

- The peak incidence of **vatha karsanam** was found in all the study group of both sex. It depends upon the control of sugar level in blood.
- The prevalence of the disease was high among middle class population 70%, 22.5%, and High class population 7.5%.
- Among dietary patterns, 60% patients consume Mixed diet with non vegetarian diet
- Regarding personal habits, 25% were smoker, 10% were Alcoholic, 5% were betalnut & Tobacco chewer,
- Out of 40 patients, 52.5%were working peoples 32.5% were house wife, 15%were other workers..
- From selected 40 patients, 17.5% comes under Kaarkaalam, 12.5% comes under Kuthirkaalam, 17.5% comes under Munpani, 20% comes under Elavenil, 32.5% comes under Mudhuvenil kaalam
- In vatham – viyanan (100%),abanan and samanam (40%), koorman (22.5%) devathathan(85%) were affected
- In pitham - Sadhaga Pitham (100%), Ranjaga pitham (25%), Aanalagam (40%), Aalosagam (22.5%) were affected.
- In Kapham - Kilethagam (40%), tharpagam (22.5%)and Santhigam (25%) were affected.
- Among Ezhu Udal Kattugal, Saaram,& seneer (100%), oon (50%) and Enbu (25%) were affected.

- Among Envagai Thervugal, naadi & sparisam (100%) Vizhi (22.5%), Niram (25%), Naa (25%), Moothiram (40%), and Malam (12.5%) were affected.
- Naadi in vatha karsanam patients felt as, Vatha pitham naadi (87.5%) and kabha vatham (12.5%)..
- In biochemical analysis the karunkali ver kudineer has iron and zinc presentation. So it shows the effect of drug in vatha karsanam.(dm neuropathy)
- The toxicity study shows the drug has no toxicity in higher doses too. So it is very safe for prolong use.
- The preclinical studies shows the drug shows significant response in both central & peripheral acting analgesic action
- The symptoms fully recovered in 70% of patients
- The HbA1C report shows good control in 62.5% of cases
- The pre clinical data were analysed statistically and observed that the drug are significant in relieving the symptoms
- The drug has easily available
- More over the drug itself has a hypoglycaemic effect and analgesic. This combination of drug should be helpful to the patient to take single drug for the disease madhumegam (diabetes) and its major complication vatha karsanam (dm neuropathy)

CONCLUSION

CONCLUSION

- The trial drug used in this study are easily available in south India
- The preparation of the trial drug is quick and easy
- The drug is like drinking water and hence the oral route of administration is easy.
- It may be noted that the drug is a safe and effective one for **vatha karsanam**
- The pharmacological study revealed that the **KARUNGALI VER KUDINEER** yielded BETTER results in rat models and there was no toxicity in the drug.
- In clinical study also there was no hypoglycemic complications were observed. Hence it could be administrated even for longer period if required.
- The pharmacological study revealed that the **KARUNGALI VER KUDINEER** yielded good results in rat models and there was no toxicity in the drug..
- The statistical analysis proved significance of clinical improvement with the treatment.
- Hence the KARUNGALI VER KUDINEER has proved its efficacy of reduces the blood sugar level and the neuropathic pain also. So the authour hope that this medicine should be a very hopeful remady for a world wide disease (diadetes) mathumegam and its major complication, vatha karsanam(dm neuropathy)

ANNEXURES

CERTIFICATES

The Tamil Nadu Dr. M.G.R. Medical University

69, Anna Salai, Guindy, Chennai-600 032

This Certificate is awarded to Dr S.UMERA

for participating as a ~~Resource Person~~ / Delegate in the VI Workshop on

"Research Methodology & Biostatistics"

for AYUSH Post-Graduates & Researchers

organized by the Department of Siddha

The Tamil Nadu Dr. M.G.R. Medical University

from 12th September 2011 to 16th September 2011

Dr. MAYILVAHANAN NATARAJAN
M.S.Orth. M.Ch.Orth. (L'pool) Ph.D. D.Sc. F.R.C.S. D.Sc. (Hon)[3]
VICE CHANCELLOR

Dr. SUDHA SESHAYYAN, M.S.
REGISTRAR (FAC)

Dr. N. KABILAN, M.D. (Siddha)
READER, DEPT. OF SIDDHA

BIOCHEMICAL ANALYSIS

ANNEXURE –I

CHEMICAL ANALYSIS OF TRIAL MEDICINES

Preparation of Sodium Carbonate extract: 2 gm of the sample is mixed 5 gm of Sodium carbonate and taken in a 100 ml beaker and 20 ml of distilled water is added. The solution is boiled for 10 minutes, cooled and then filtered. The filtrate is called sodium carbonate extract.

S.No.	Experiment	Inference	
		Drug	**Drug**
1	Test for Acid Radicals		
a.	**Test for Sulphate** 2 ml of the above prepared extract is taken in a test tube. To this add 2ml of 4% Ammonium oxalate solution.	Absence of White Precipitate	Absent
b.	2ml of extract is added with 2ml of dilute hydrochloric acid until the effervescence ceases off. Then 2ml barium chloride solution is added.	Absence of White Precipitate	Absent
2.	**Test for Chloride:** 2ml of extract is added with dilute nitric acid till the effervescence ceases. Then 2ml of silver nitrate solution is added.	white precipitate is developed.	Present

3.	**Test for Phosphate** 2ml of the extract is treated with 2 ml of Ammonium molybdate solution and 2ml of concentrated nitric acid.	Yellow Precipitate is obtained.	Absent
4.	**Test for Carbonate:** 2ml of the extract is treated with 2ml of magnesium sulphate solution.	Absence of white precipitate	Absent
5.	**Test for Sulphide:** 1 gm of the substance is treated with 2ml of concentrated Hydrochloric acid	Absence of Rotten egg smelling	Absent
6.	**Test for Nitrate:** 1gm of the substance is heated with copper turnings and concentrated sulphuric acid and viewed the test tube vertically down.	Absence of reddish brown gas.	Absent
7. a.	**Test for Fluoride and oxalate** 2ml of the extract is added with 2ml of dilute acetic acid and 2ml of calcium chloride solution and heated.	Absence of white precipitate	Absent
b.	5 drops of clear solution is added with 2ml of dilute sulphuric acid and slightly warmed to this, 1 ml of dilute potassium permanganate solution is added.	Absence of KMNO4 solution discolourisation.	Absent

8.	**Test for Nitrite** 3 drops of the extract is placed on a filter paper. On that, 2 drops a Acetic Acid and 2 drops of Benzidine solution is placed.	Absence of yellowish red colour	Absent
9.	**Test for Borate** 2 pinches of the substance is made into paste by using Sulphuric acid and Alcohol (95%) and introduced into the blue flame.	Absence of Green tinged flame	Absent
II.	**TEST FOR BASIC RADICALS**		
10.	**Test for lead** 2 ml of the extract is added with 2 ml of Potassium iodide solution	Absence of Yellow precipitate	Absent
11a	**Test for Copper** One pinch of substance is made into paste with concentrated Hydrochloric acid in a watch glass and introduced into the non luminous part of the flame.	Bluish green coloured flame is obtained.	Absent
b.	2ml of the extract is added with excess of Ammonia solution	Absence of deep blue	Absent
12.	**Test for Aluminium** To the 2 ml of extract. Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent
13a	**Test for Iron** To the 2 ml of extract, 2 ml of Ammonium Thiocyanate solution is added.	Blood red colour is obtained.	Present
b.	To the 2 ml of extract, 2 ml of Ammonium Thiocyanate solution and 2 ml of concentrated Nitric Acid is added.	Blood red colour is obtained.	present
14.	**Test for Zinc** To the 2 ml of extract Sodium Hydroxide solution is added in drops to excess.	White precipitate is obtained	Present
15.	**Test for Calcium** 2 ml of the extract is added with 2 ml of 4% Ammonium Oxalate solution.	Absence of White precipitate.	Absent
16.	**Test for Magnesium** 2ml of extract, Sodium Hydroxide solution is added in drops to excess.	Absence of White precipitate.	Absent

17.	**Test for Ammonium** 2 ml of extract few ml of Nessler's Reagent and excess of Sodium Hydroxide solution are added.	Absence of Reddish brown precipitate	Absent
18.	**Test for Potassium** A pinch of substance is treated with 2 ml of Sodium Nitrite solution and then treated with 2 ml of Cobal Nitrate in 30% glacial Acetic acid.	Absence of Yellow precipitate	Absent
19.	**Test for Sodium** 2 pinches of the substance is made into paste by using Hydrochloric acid and introduced into the blue flame.	Absence of Yellow colour flame	Absent
20.	**Test for Mercury** 2 ml of the extract is treated with 2 ml of Sodium Hydroxide solution.	Absence of yellow precipitate	Absent
21.	**Test for Arsenic** 2 ml of extract is treated with 2 ml of silver Nitrate solution	Absence of Yellow precipitate.	Absent
22.	**Test for Starch** 2ml of extract is treated with weak iodine solution	Absence of Blue colour	Absent
23.	**Test of reducing Sugar** 5ml of Benedicts qualitative solution is taken in a test tube and allowed to boil for 2 minutes and added 10 drops of the extract and again boiled for 2 minutes. The colour changes are noted.	Green colour is obtained.	Present
24.	**Test of the alkaliods** 2ml of the extract is treated with 2ml of potassium iodide solution	Red colour developed	Present
25.	**Test for proteins: (biuret test)** Take 2 ml of solution and 2ml of 5% sodium hydroxide, mix and add 2 drops of copper sulphate solution.	Absence of violet colour	Absent

RESULTS:

The given sample contains.
Drug- karungali ver kudineer – 1gm
Chemicals present:
Chloride
Iron
Zinc
Reducing sugar
alkaloides
.

TOXICOLOGICAL STUDY

ANNEXURE II

ACUTE AND SUB ACUTE TOXICITY STUDY ON KARUNGALI VER KUDINEER

Animals

Mice of either sex weighing 25-30g and rats weighing 210-240g were obtained from the animal house of Vels University. The animals were used with the approval of the Institute animal ethics committee and obtained from Vels University, Chennai. They were fed with a balanced standard pellet diet and maintained under standard laboratory conditions, providing 24-28^{0}C temperature, standard light cycle (12 h light, 12 h dark) and water ad libitum.

Animals were kept in cages with raised floors of wide mesh to prevent coprophagy. Animal welfare guidelines were observed during the maintenance period and experimentation. The rats were randomly assigned to control and different treatment groups, six animals per group. The animals were acclimatized for one week under laboratory conditions.

ACUTE TOXICITY STUDY-OECD 425 GUIDELINES

Acute oral toxicity test for the Karungali Ver Kudineer was carried out as per OECD Guidelines 425. As with other sequential test designs, care was taken to ensure that animals are available in the appropriate size and age range for the entire study. The test substance is administered in a single dose by gavage using a stomach tube or a suitable intubation cannula. The fasted body weight of each animal is determined and the dose is calculated according to the body weight. After the substance has been administered, food was withheld for a further 2 hours in mice.

The animals were observed continuously for the first 4 h and then each hour for the next 24 h and at 6 hourly intervals for the following 48 h after administering of the test drug, to observe any death or changes in general behaviour and other

physiological activities. Single animals are dosed in sequence usually at 48 h intervals. However, the time interval between dosing is determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the next dose was delayed until one is confident of survival of the previously dosed animal. General behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes, change in skin and fur, mortality and the body weight changes were monitored daily. The time of onset, intensity, and duration of these signs, if any, was recorded.

SUB-ACUTE TOXICITY

In a 28-days sub acute toxicity study, twenty four either sex rats were divided into four groups of 6 rats each. Group I that served as normal control was administered with distilled water (p.o.) while groups II, III and IV were administered daily with the Karungali Ver Kudineer (p.o.) for 28 days at a dose of 2, 4 and 8ml/kg respectively. The animals were then observed daily for gross behavioural changes and any other signs of subacute toxicity.

The weight of each rat was recorded on day 0 and weekly throughout the course of the study, food and water consumption per rat was calculated. At the end of the 28 days they were fasted overnight, each animal was anaesthetized with diethylether, following which they were then dissected and blood samples were obtained by cardiac puncture into heparinised tubes. The blood sample collected from each rat was centrifuged with 3000 X g at 4^{o}C for 10 min to separate the serum and used for the biochemical assays.

Hematological and blood biochemical analyses

After 4 weeks of the once daily treatment of Karungali Ver Kudineer, all animals were kept fasted for 16-18 h and then anesthetized with anesthetic ether on the 28th day. Blood samples for hematological and blood chemical analyses were taken from retro orbital vein. Heparinized blood samples were taken for determining complete blood count (white blood cell count, differential white blood cell count,

platelet count, red blood cell count, hematocrit, and hemoglobin) by semiautomated hematology analyzer.

The serum from non-heparinized blood was carefully collected for blood chemistry and enzyme analysis (glucose, creatinine, total protein, albumin, total and direct bilirubins, serum glutamate-oxaloacetate transaminase (SGOT), serum glutamate pyruvate transaminase (SGPT), and alkaline phosphatase (ALP)) were automatically determined using autoanalyzer.

Necropsy

All rats were sacrificed after the blood collection. The positions, shapes, sizes and colors of internal organs were evaluated. The Spleen, Testes, Pancrea, Lung, Liver, Brain, Heart, Stomach, Intestine, Bone, Ovary, and Kidney tissues were excised from all rats to visually detect gross lesions, and weighed to determine relative organs' weights and preserved in 10% neutral formalin for histopathological assessment. The tissues were embedded in paraffin, and then sectioned, stained with haematoxylin and eosin and were examined microscopically.

Statistical analysis

Values were represented as mean ± SEM. Data were analyzed using one-way analysis of variance (ANOVA) and group means were compared using the Tukey-Kramer Multiple Comparison test using Graph Pad Instat-V3 software. $P < 0.05$ was considered significant.

RESULTS

All the animals from control and all the treated dose groups up to 400 mg/kg survived throughout the dosing period of 28 days. No signs of significant intoxication were observed in animals from lower to higher dose groups during the dosing period of 28 days. Animals from all the treated dose groups exhibited comparable body weight gain with that of controls throughout the dosing period of 28 days.

Food consumption of control and treated animals was found to be comparable throughout the dosing period of 28days. Ophthalmoscopic examination, conducted prior to and at the end of dosing period on animals from control and all the treated dose groups did not reveal any abnormality. The results of haematological investigations revealed following no significant changes in the values of different parameters investigated when compared with those of respective controls; However, the increase or decrease in the values obtained was within normal biological and laboratory limits.

Results of Biochemical investigations revealed no statistically significant changes when compared with those of control. Functional observation tests conducted at termination revealed no abnormalities. Urine analysis, conducted at the end of the dosing period in week 4 and at the end of recovery period in week 6, revealed no abnormality attributable to the treatment. Organ weight data of animals sacrificed at the end of the dosing period was found to be comparable with that of respective controls. Gross pathological examination did not reveal any abnormality. Histopathological examination did not reveal any abnormality.

CONCLUSION

In the present toxicological investigation, no toxic effect was identified upto 8ml/kg of Karungali Ver Kudineer administered through oral route for 28 days. So, it can be concluded that the Karungali Ver Kudineer can be used for therapeutic use in human with the dosage recommendations of upto 8ml/kg body weight p.o.

Table 1: Dose finding experiment and its behavioral Signs of Toxicity

No	Dose ml/kg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1.	**5**	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2.	**10**	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3.	**20**	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7. Decreased Motor Activity 8. Tremors 9. Convulsions 10. Muscle Spasm 11. Catatonia 12. Muscle relaxant 13. Hypnosis 14. Analgesia 15.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality

Table 2. Body wt (g) of rats exposed to Karungali ver Kudineer for 28days.

Dose (ml/kg/day)	Days				
	1	**7**	**14**	**21**	**28**
Control	121.41±5.40	122.22±4.62	124.15±5.00	132.15±4.72	135.41±4.00
2	125.44±4.28	127.20±5.00	130.18±5.11	133.40±5.10	135.14±4.45
4	128.62±5.12	128.17±4.52	130.21±5.00	132.01±5.02	134.28±5.00
8	121.18±5.20	125.12±5.20	130.14±5.21	132.00±4.78	135.22±5.12

Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}P>0.05$; Vs Control N=6.

Table 3. Food (g/day) intake of rats exposed to Karungali ver Kudineer for 28days.

Dose (ml/kg/day)	Days (gms/rats)				
	1	**7**	**14**	**21**	**28**
Control	53.14±2.28	53.44±2.44	55.12±2.11	52.13±2.61	51.15±3.40
2	54.82±2.12	55.23±2.42	52.42±2.60	55.18±2.45	56.72±3.12
4	55.13±2.42	52.25±2.30	57.47±2.35	52.46±2.10	51.24±3.16
8	52.11±2.40	53.40±2.41	59.40±2.22	54.14±2.12	50.10±3.12

Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}P>0.05$; Vs Control N=6.

Table 4. Water intake of rats exposed to Karungali ver Kudineer for 28days.

Dose (ml/kg/day)	Days(ml/rat)				
	1	**7**	**14**	**21**	**28**
Control	50.00±2.52	50.25±3.00	51.16±2.88	50.62±3.00	52.15±3.16
2	50.25±2.34	51.24±3.15	50.56±3.44	48.21±3.40	50.25±2.28
4	51.40±2.25	50.51±3.45	50.25±3.42	48.13±2.48	52.48±3.45
8	50.44±3.42	50.40±3.10	55.28±3.25	52.40±3.20	54.44±3.36

Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}P>0.05$; Vs Control N=6.

Table 5. Hematological parameters after 28days treatment with Karungali ver Kudineer.

Parameter	Control	2ml/kg	4ml/kg	8ml/kg
RBC (mm^3)	7.20±0.33	7.25±0.31	7.18±0.24	7.22±0.28
HB (%)	14.62±0.27	14.55±0.23	14.72±0.25	14.66±0.32
Leukocyte ($x10^6$/mL)	10.14±1.22	10.12±1.25	10.14±1.24	10.52±1.25
Platelets ($X10^5$/µl)	1.33±0.15	1.30±0.14	1.32±0.12	1.30±0.14
MCV (g/l)	85.02±4.0	85.00±5.0	85.10±4.15	84.88±5.12
Neutrophil (%)	52.12±3.2	52.18 ±3.4	51.52±3.2	52.11±3.0
Lymphocytes (%)	44.14±2.22	45.10±3.0	45.25±2.8	45.21±3.2
Eosinophil's (%)	5.0±0.4	5.0±0.4	5±0.3	5±0.3
Monocytes (%)	3.0±0.02	3.0±0.03	3.0±0.03	3.0±0.02
Basophils (%)	0±0	0±0	0±0	0±0
ESR(mm)	1±00	1±00	1±00	1±00
PCV	45.42±3.12	44.00±3.33	43.00±3.21	43.26±3.15

Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}P>0.05$; Vs Control N=6.

Table 6. Effect of treatment with Karungali ver Kudineer biochemical parameters.

Dose (mg/kg)	Control	2ml/kg	4ml/kg	8ml/kg
Total Bilirubin (mg/dL)	0.211±0.06	0.214±0.04	0.215±0.05	0.215±0.05
Bilirubin direct (mg/dL)	0.1±0.04	0.1±0.05	0.1±0.04	0.1±0.05
ALP (U/L)	71.04±2.5	71.21±2.8	71.10±3.0	70.28±2.5
SGOT (U/L)	74.10±3.2	74.00±3.1	73.11±3.8	74.22 ± 3.4
SGPT(U/L)	81.1±3.0	82.00±3.2	81.50±2.5	80.14±2.9
Total Protein(g/dl)	9.00±1.22	9.10±0.23	8.15±0.22	8.21±0.25
Albumin(g/dl)	3.13±0.20	3.41±0.22	3.40±0.31	3.12±0.30
Globulin(g/dl)	5.00±0.28	4.78±0.26	4.90±0.24	4.88±0.26
Urea (mg/dL)	54.40±1.35	54.00±3.00	54.08±2.88	55.01±2.73
Creatinine (mg/dL)	29.56±3.4	29.11±3.0	28.04±3.14	27.22 ± 3.2
Uric acid (mg/dL)	1.6±0.18	1.5±0.16	1.7±0.12	1.6±0.15
Na m.mol	142.42±4.20	142.21±3.00	142.14±3.56	142.00±3.02
K m.mol	20.02±2.22	19.00±2.19	20.11±2.30	20.18±2.46
Cl m.mol	102.35±4.88	101.36±5.41	102.60±5.92	101.40±4.00

Values are mean ± S.E.M. (Dunnet't' test). $^{ns}P>0.05$ Vs Control

Table-8. Lipid Profile

Dose (mg/kg)	Control	2ml/kg	4ml/kg	8ml/kg
Total cholesterol (mg/dL)	42.11±2.45	40.27±2.66	41.14±2.64	41.89±2.79
HDL(mg/dL)	14.22±2.28	14.42±1.84	14.11±1.82	14.00±2.36
LDL(mg/dL)	42.12±2.62	42.05±3.00	42.55±3.00	42.32±3.24
VLDL(mg/dl)	16.25±2.33	16.18±2.42	16.22±1.34	15.19±1.20
Triglycerides (mg/dl)	85.12±2.49	85.21±2.34	85.02±3.04	85.00±2.45
Blood glucose(mg/dl)	125.23±3.00	125.72±3.12	125.00±3.05	125.82±2.42

Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}P>0.05$; Vs Control N=6.

Table-9 Urine Analysis

Parameters	**Control**	**2ml/kg**	**4ml/kg**	**8ml/kg**
Colour	Yellow	Yellow	Yellow	Yellow
Transparency	Clear	Slightly turbid	Slightly cloudy	Slightly turbid
Specific gravity	1.010	1.010	1.010	1.010
PH	>7.2	>8.0	>8.0	>9.0
Protein	Nil	3+	3+	3+
Glucose	Nil	Nil	Nil	Nil
Bilirubin	-ve	-ve	-ve	-ve
Ketones	-ve	+ve	+ve	+ve
Blood	Absent	Absent	Absent	Absent
Urobilinogen	Normal	Abnormal	Abnormal	Abnormal
Pus cells	0-cells/HPF	1-cell/HPF	2-cells/HPF	1-cell/HPF
RBCs	Nil	Nil	0-1cells/HPF	Nil
Epithelial cells	Nil	1-cell/HPF	Nil	1-cell/HPF
Crystals	Nil	Nil	Nil	Nil
Casts	Nil	Nil	Nil	Nil
Others	Bacteria seen	Bacteria seen	Bacteria seen	Bacteria seen

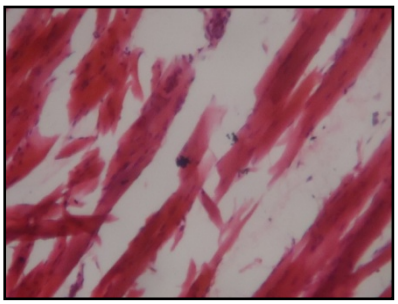
Table 10. Effect of Karungali ver Kudineer on organ weight

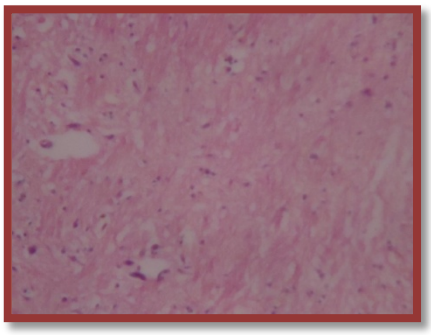
Dose (mg/kg)	Control	2ml/kg	4ml/kg	8ml/kg
Liver (g)	7.00±0.10	6.98±0.12	6.90±0.14	7.10±0.15
Heart (g)	0.62±0.05	0.62±0.05	0.61±0.06	0.63±0.05
Lung (g)	1.41±0.12	1.42±0.12	1.39±0.11	1.40±0.12
Spleen (g)	0.64±0.06	0.68±0.05	0.67±0.05	0.68±0.06
Ovary (g)	1.65±0.12	1.66±0.10	1.65±0.12	1.64±0.14
Testes (g)	1.40±0.14	1.42±0.11	1.43±0.13	1.44±0.12
Brain (g)	1.55±0.12	1.56±0.14	1.55±0.11	1.54±0.12
Kidney (g)	0.70±0.05	0.72±0.05	0.71±0.04	0.72±0.05
Stomach (g)	1.32±0.15	1.31±0.12	1.32±0.10	1.32±0.15

Values are mean ± S.E.M. (Dunnet 't' test). $^{ns}P>0.05$; Vs Control N=6.

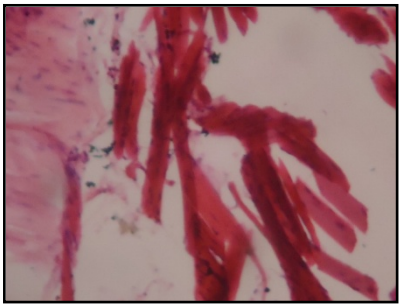
BONES BRAIN

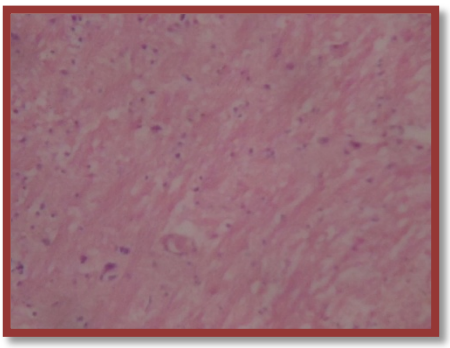
MINIMAL DOSAGE (2ml)



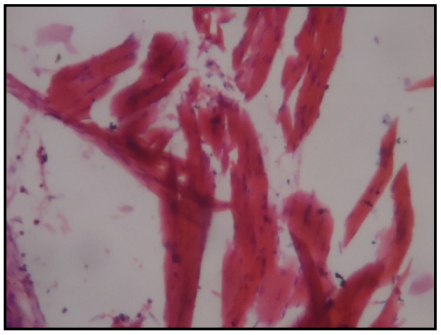


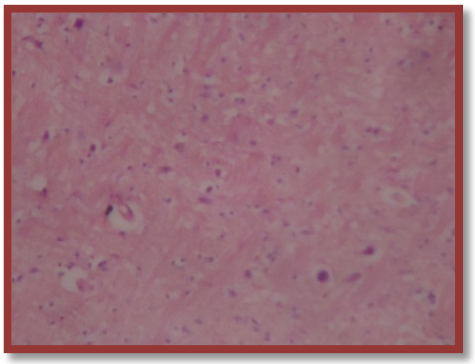
MODARATE DOSAGE (4ml)





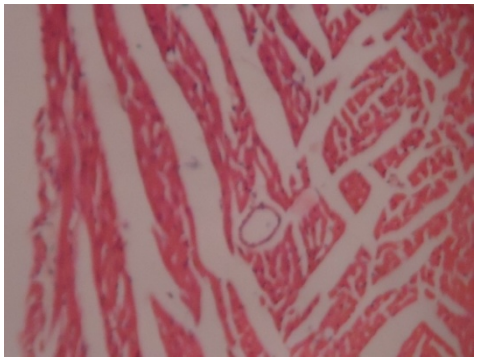
HIGH DOSAGE (8ml)

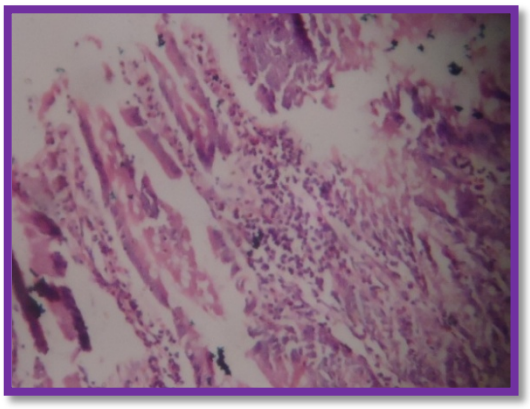




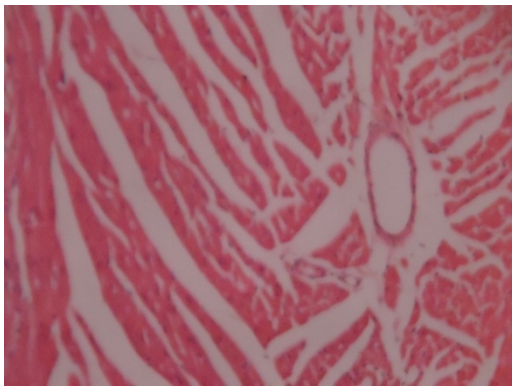
HEART INTESTINES

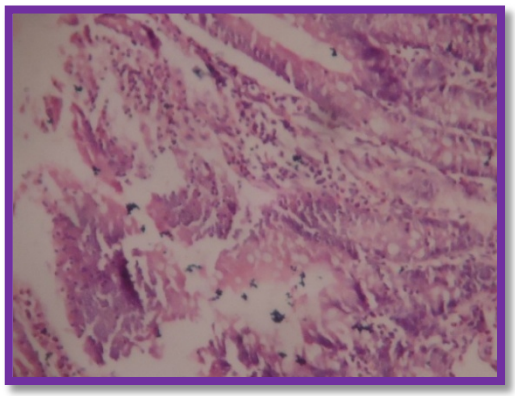
MILD DOSE (2ml)



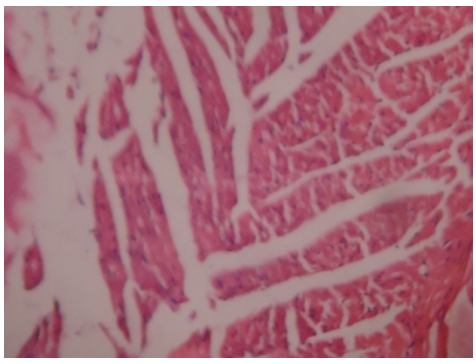


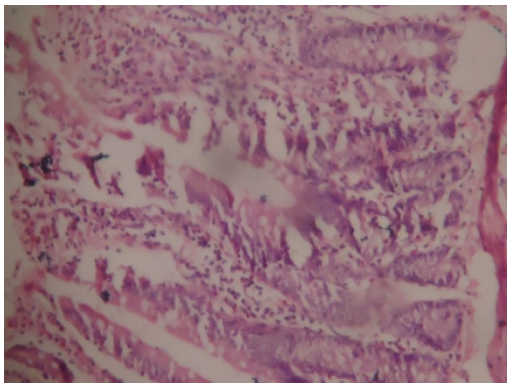
MODARATE DOSAGE (4ml)





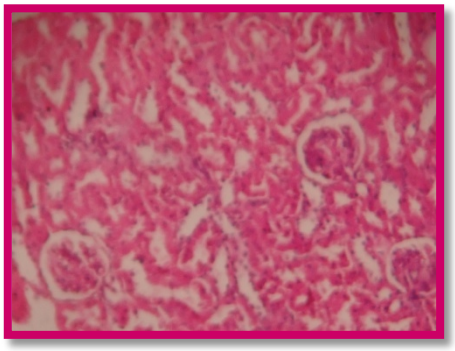
HIGH DOSAGE (8ml)

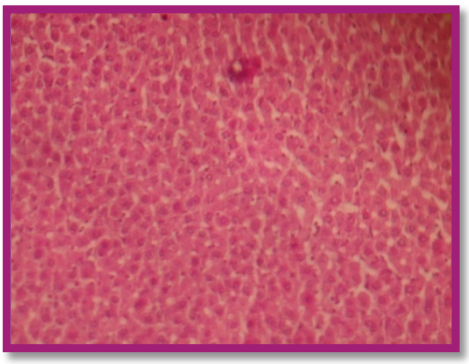




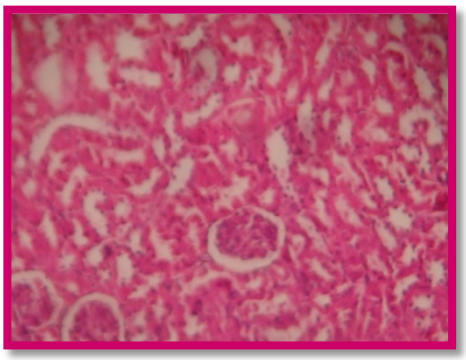
KIDNEY LIVER

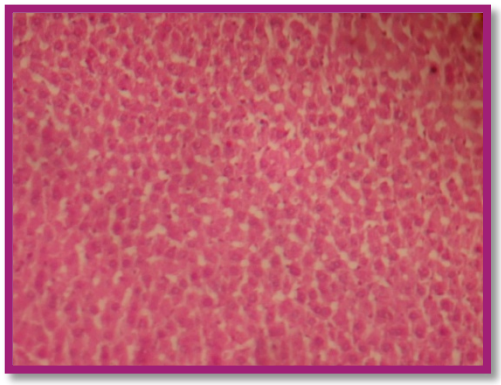
MILD DOSE (2ml)



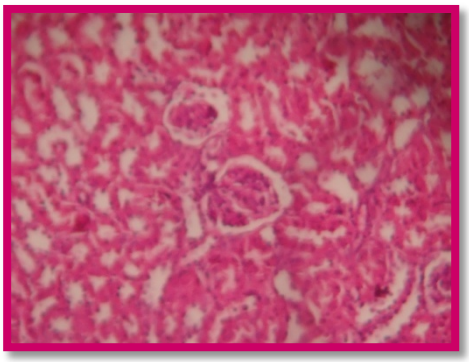


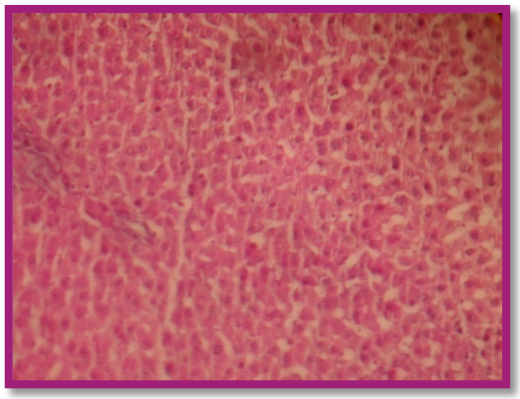
MODARATE DOSE (4ml)





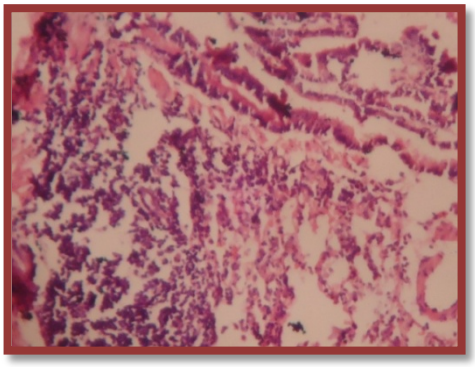
HIGH DOSE (8ml)

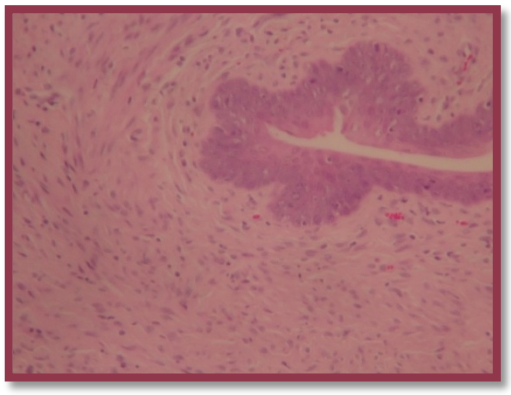




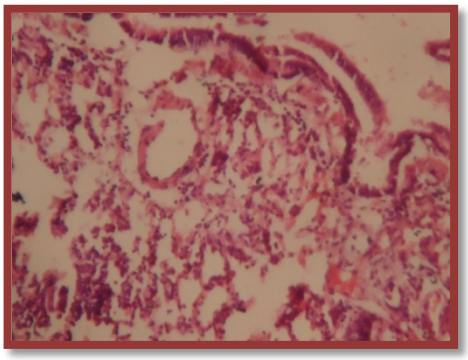
LUNGS OVARIES

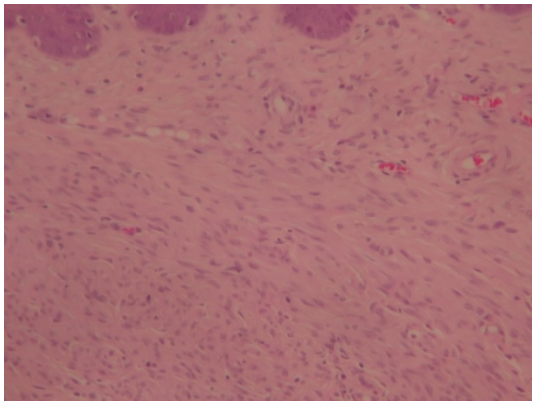
MILD DOSAGE (2ml)



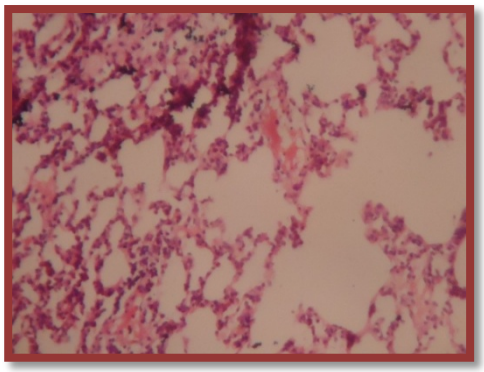


MODARATE DOSAGE (4ml

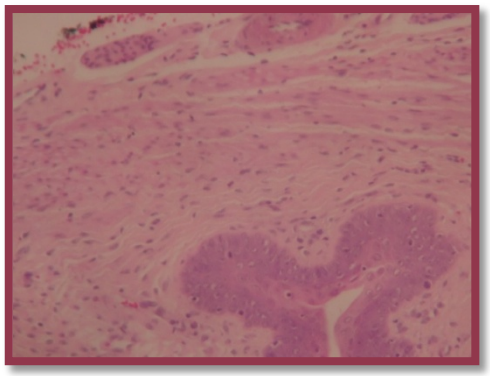




HIGH DOSE (8 ml)

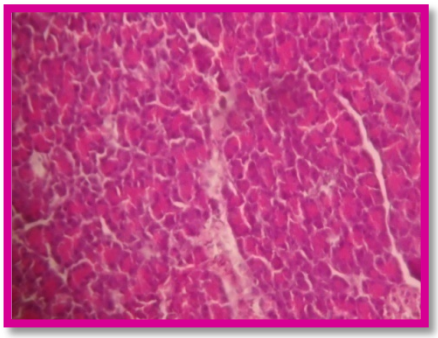


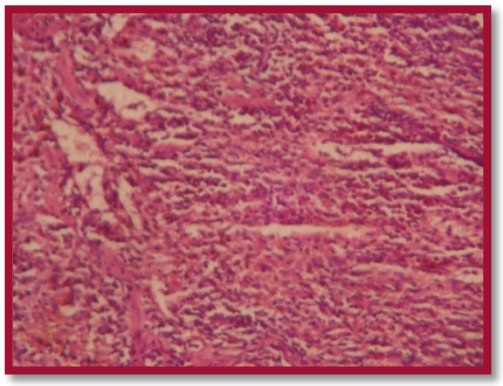
PANCREAS



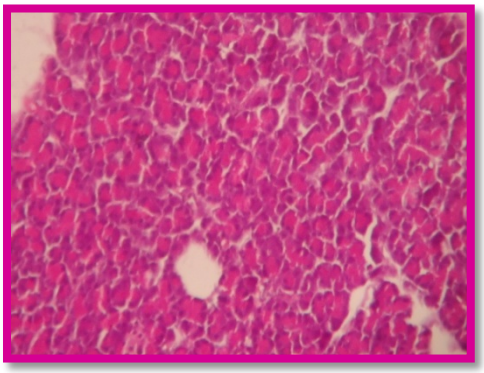
STOMACH

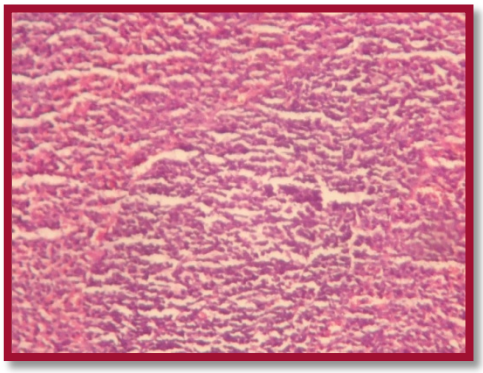
MILD DOSE (2ml)



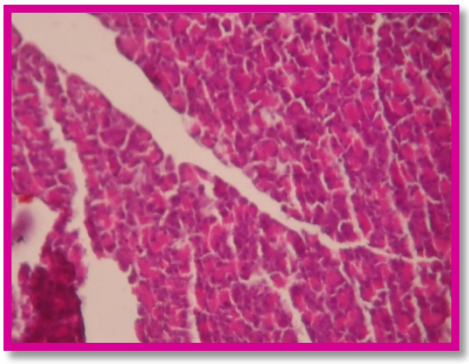


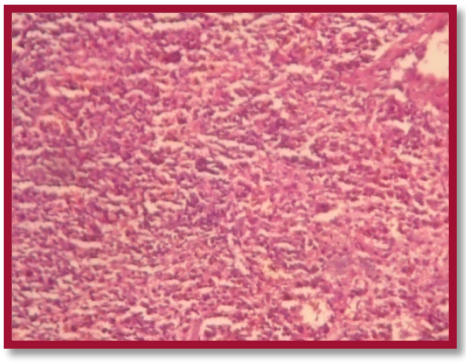
MODARATE DOSE (4 ml)





HIGH DOSE (8ml)

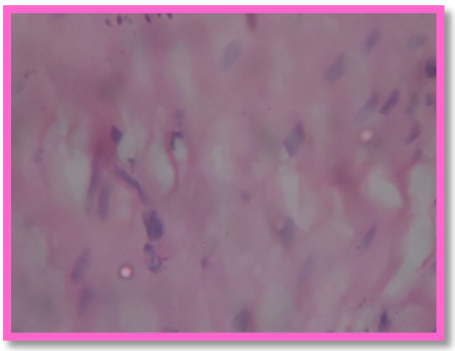


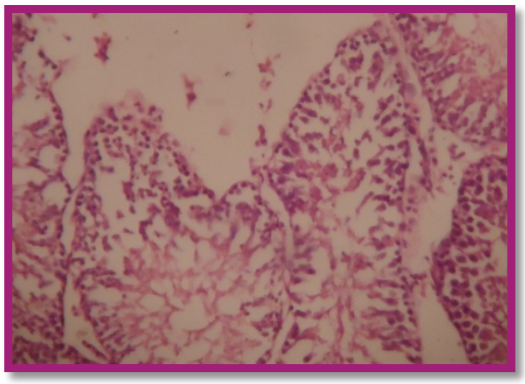


SPLEEN

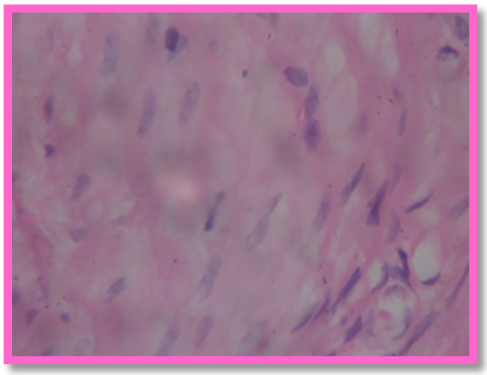
TESTIS

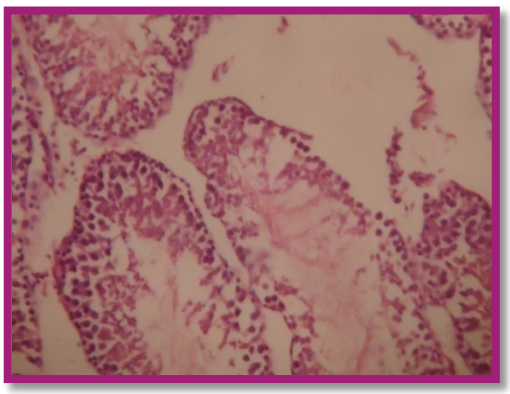
MILD DOSE (2ml)



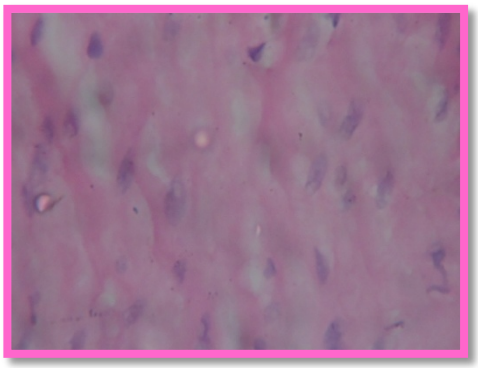


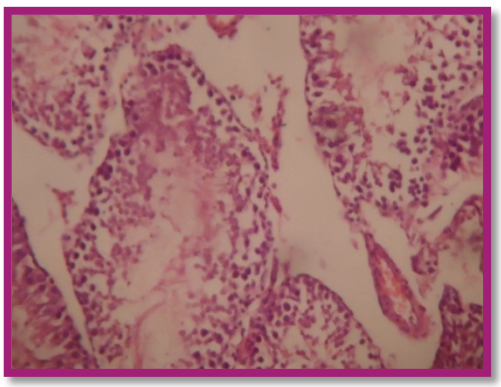
MODARATE DOSE (4ml)





HIGH DOSE (8ml)





PHARMACOLOGICAL STUDY

ANNEXURE III

EVALUATION OF ANALGESIC EFFECT OF KARUNGALI VER KUDINEER

INTRODUCTION

Pain is a general problem of people throughout the world. Pain is a primary response of body injury, inflammation, cancer etc. It can also occur for brain or nerve injury. With many pathological conditions, tissue injury is the immediate cause of the pain, and this result in the local release of a variety of chemical agents which act on the nerve terminals, either activating them directly or enhancing their sensitivity to other forms of stimulation. Many plant based traditional siddha medicines propose a rich medicinal property used by the population for the treatment of different types of pain. However, there were not enough scientific investigations on the analgesic activities conferred. One of such drug from Indian system of medicine is Karungali ver is an indigenous herb and has been used in for treating rheumatism and neuropathic painful conditions. The drug is employed in different forms such as decoction, syrup, infusion and powder (Chooranam). Since no scientific data are available to justify the traditional analgesic potentials of this drug.

Hence, the present study was planned to validate the therapeutic use in treatment of painful conditions. Analgesic therapy is dominated by two major classes of analgesic drugs; namely opioids and non steroidal anti-inflammatory drugs (NSAIDs). Both classes of analgesic drugs produce serious side effects, such as gastrointestinal disturbances, renal damages (with NSAIDs drugs), respiratory depression and possibly dependence (with opioids). The ultimate aim of the present study was to find out the safety and efficacy of the Karungali ver Kudineer which has been traditionally used to treat pain. This study was intended to evaluate the peripheral and central analgesic activity of Karungali ver Kudineer in experimental animal models following oral administration.

MATERIALS AND METHODS

Drugs and chemicals

Acetic acid, and CMC, all from Sigma-Aldrich Chemicals were the chemicals used. The standard drugs aspirin and Pentazocine was procured from the local market. All the other chemicals and drugs used were of analytical grade.

Stock solution preparation

As recommended in standard siddha literature, the powder form of well dried 100grams of Karunkali Ver was mixed with 200ml of distilled water. Then this was boiled continuously till the total volume is concise to $1/4^{th}$ as a decoction. The filtered supernatant fluid was employed for the preclinical study.

Animals

Albino mice (22–28 g) either sex were obtained from the animal house of animal housing facility of department of pharmacology, Vels University, Chennai. Animals were maintained at standard laboratory conditions and fed with standard feeding pellets (Sai durga foods, Bangalore). Prior to treatment, the animals were fasted for 10 and 12 h respectively. However, water was made available ad libitum. (Approval number: XIII/VELS/PCOL/05/2000/CPCSEA/IAEC/08.08.2012).

Experimental Methods

Acute toxicity safety Study

Acute oral toxicity test for the Karungaliver Kudineer was carried out as per OECD Guidelines 425. As with other sequential test designs, care was taken to ensure that animals are available in the appropriate size and age range for the entire study. The test substance is administered in a single dose by gavage using a stomach tube or a suitable intubation cannula. The fasted body weight of each animal is determined and the dose is calculated according to the body weight. After the substance has been administered, food was withheld for a further 2 hours in mice. The animals were observed continuously for the first 4 h and then each hour for the next 24 h and at 6 hourly intervals for the following 48 h after administering of the test drug, to observe

any death or changes in general behaviour and other physiological activities. Single animals are dosed in sequence usually at 48 h intervals.

However, the time interval between dosing is determined by the onset, duration, and severity of toxic signs. Treatment of an animal at the next dose was delayed until one is confident of survival of the previously dosed animal. General behavior, respiratory pattern, cardiovascular signs, motor activities, reflexes, change in skin and fur, mortality and the body weight changes were monitored daily. The time of onset, intensity, and duration of these signs, if any, was recorded.

Evaluation of analgesic activity by Eddy's Hotplate method

The hot-plate test method was employed to assess the analgesic activity. The temperature of the cylinder was set at 55±0.5^{0}C. The experimental mice were divided into four groups. Each mouse acted as its own control. Prior to treatment, the reaction time of each mouse (licking of the forepaws or jumping response) was done at 0 and 10min interval. The average of the two readings was obtained as the initial reaction time. The reaction time following the administration of the Karungaliver Kudineer (1, 2, 4ml/kg, p.o.), Pentazocine (5mg/kg) and Saline (p.o.), was measured at 30, 60, 90 and 120 minutes after a latency period of 30 mins. The Percentage analgesic activity was calculated.

Antinociceptive testing

The antinociceptive property of Karungaliver Kudineer was tested using the model of writhing response in mice. Swiss albino mice of either sexes weighing 20-30 g were used. The writhing syndrome was elicited by an intraperitoneal injection of 0.7% acetic acid at the dose of 0.1ml/10 g body weight. For the test group of animals Karungaliver Kudineer at the dose level of 1, 2, 4ml/kg, p.o. and for control group vehicle saline and Aspirin 100mg/kg was orally administered into the mice 30 min before acetic acid and the number of writhes was noted for 15 min beginning 5 min after acetic acid injection.

Statistical data

Data were presented as mean ± S.E.M. Statistical differences between control and treated groups were tested by one way ANOVA followed by dunnet's test.

RESULTS AND DISCUSSION

Karungaliver Kudineer was found safe at all test doses (5, 10 and 20ml/kg p.0.). During 24h assessment time, test animals were found normal. Hence the therapeutic dose was fixed as 2 and 4ml/kg according to the safety guidelines. Acetic acid, which is used as an inducer for writhing syndrome, causes algesia by liberation of endogenous substances, which then excite the pain nerve endings. It was found that Aspirin caused an effective or significant inhibition on the writhing response induced by acetic acid.

Doses of 1, 2 and 4ml/kg of the Karungaliver Kudineer were evaluated to verify the peripheral analgesic effect. But the results for the animal group treated with Karungaliver Kudineer did not differ significantly from negative control. Hence, it is assumed that Karungaliver Kudineer has no statistically significant peripheral analgesic effect. Therefore, despite of the mild effect observed for the doses of 4ml/kg for this test, it was not statistically significant to that of control.

Similarly, the result of the analgesic activity evaluated using hot plate method revealed that the reaction time for mice was significantly increased in a dose dependent manner after 90minutes of oral administration. The Karungaliver Kudineer at the both 2&4ml/kg doses remarkably protected the mice against thermally induced noxious stimuli, which was evidenced from the hot plate test. Hot plate test was assayed to characterize the central analgesic activity. The results showed that the pain relief was achieved in a dose dependent manner, at both test doses (1, 2 and 4ml/kg).

It is known that non-steroidal anti-inflammatory drugs usually do not increase the pain threshold in normal tissues, whereas local anesthetics and narcotics do. The acetic acid induced abdominal writhes were observed to be 40.44 ± 6.90 over the period of 10 min in the control. The number of abdominal writhes were not significantly ($p>0.05$) inhibited by Karungaliver Kudineer. Standard drug, acetyl salicylic acid significantly inhibited writhes by about 73.04% over the control. The

pain protective effect exerted by the Karungaliver Kudineer on the mouse by hot plate method, suggest that the analgesic effect of the drug may be centrally mediated but not peripherally. Acetic acid-induced writhing is a well recommended protocol in evaluating medicinal agents for their analgesic property. The pain induction caused by liberating endogenous substances as well as some other pain mediators such as arachidonic acid via cyclooxygenase, and prostaglandin biosynthesis.

This pain paradigm is widely used for the assessment of peripheral analgesic activity due to its sensitivity and response to the compounds at a dose which is not effective in other methods. The local peritoneal receptor could be the cause of abdominal writhings. Pain sensation in acetic acid induced writhing paradigm is elicited by producing localized inflammatory response due to release of free arachidonic acid from tissue phospholipids via cyclo-oxygenase (COX), and producing prostaglandin specifically PGE2 and PGF2α, the level of lipoxygenase products may also increases in peritoneal fluids. These prostaglandin and lipoxygenase products cause pain by increasing capillary permeability. The substance inhibiting the writhings will have analgesic effect preferably by inhibition of prostaglandin synthesis, a peripheral mechanism of pain inhibition. Thermal nociception models such as hot plate test used to evaluate central analgesic activity.

CONCLUSION

Karungaliver Kudineer showed significant ($P<0.01$) analgesic effect in the hot plate test, implicating supraspinal analgesic pathways. In these pain paradigms pentazocin, which is similar to the action of opioid agonists (e.g. morphine), raised the pain threshold level within 30min of administration. Karungaliver Kudineer showed maximum analgesic effect after 90min of administration. In conclusion, the Karungaliver Kudineer was proved as a safe siddha remedy for the treatment of algesia at the dose level of 4ml/kg body weight orally.

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Table 1: Dose finding experiment and its behavioral Signs of Toxicity

No	Dose ml/kg	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20
1.	**5**	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
2.	**10**	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-
3.	**20**	+	-	-	+	-	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-

1. Alertness 2. Aggressiveness 3. Pile erection 4. Grooming 5. Gripping 6. Touch Response 7. Decreased Motor Activity 8. Tremors 9. Convulsions 10. Muscle Spasm 11. Catatonia 12. Muscle relaxant 13. Hypnosis 14. Analgesia 15.Lacrimation 16. Exophthalmos 17. Diarrhoea 18. Writhing 19. Respiration 20. Mortality

Table 2: Effect of Karungaliver Kudineer **on pain induced by hot plate method**

Treatment	Dose	Reaction time in sec. before drug	% increase in reaction time after drug treatment			
			30min	60min	90min	120min
Control	Saline 2ml/kg	3.2±0.05	8.1±0.02	12.6±0.5	15.48±0.6	15.30±0.5
Karungaliver Kudineer	1mL/kg	3.2±0.04	19.6±0.32**	25.42±1.18**	37.25±2.23**	35.38±1.12**
Karungaliver Kudineer	2mL/kg	3.1±0.05	27.2±0.30**	36.20±1.42**	55.39±2.81**	46.20±1.20**
Karungaliver Kudineer	4mL/kg	2.4±0.05	32.4±0.26**	39.56±1.33**	64.00±2.48**	53.18±1.20**
Pentazocine	5mg/kg	3.0±0.04	54.1±1.33**	65.72±2.88**	69.10±2.45**	66.04±2.00**

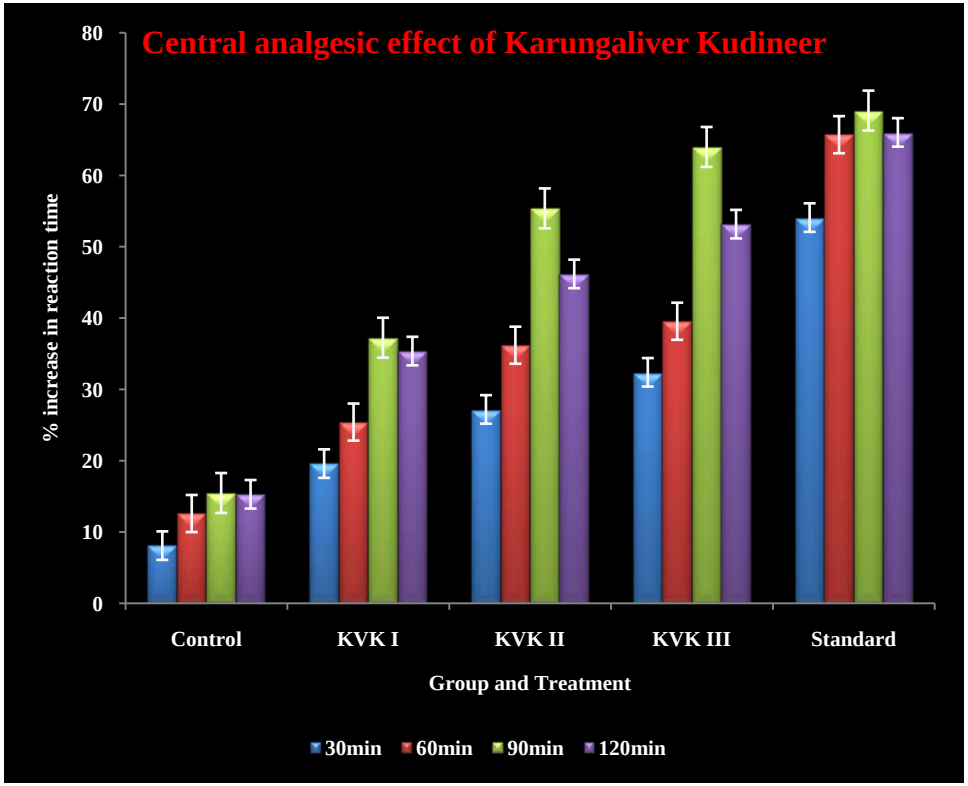
Values expressed in mean ±SEM, Significant **P<0.01 (n=6)

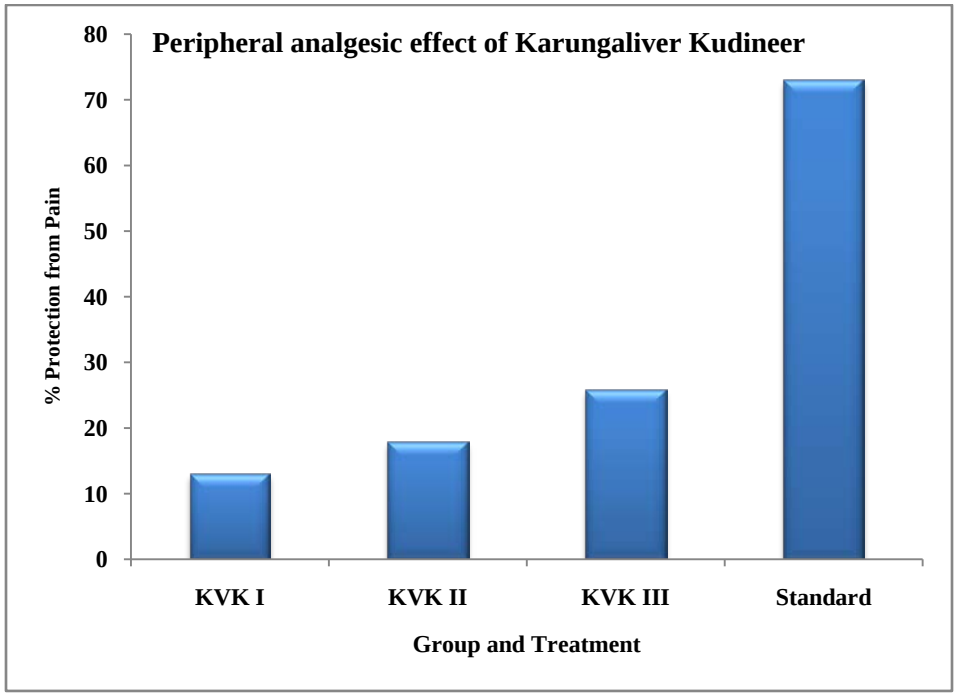
Table –3. Effect of Karungaliver Kudineer on writhing response in mice

Treatment	**Dose (mg/kg)**	Number of writhes	Inhibition (%)
Control	Saline 2ml/kg	40.44±6.9	-----
Karungaliver Kudineer	1mL/kg	35.18±5.67	13.00
Karungaliver Kudineer	2mL/kg	33.24±4.00	17.80
Karungaliver Kudineer	4mL/kg	30.01±2.82	25.79
Acetyl salicylic acid	100mg/kg	10.9±3.00**	73.04

Values are expressed as Mean±S.E.M. Drug and test compounds were given orally 30 min before 0.3% acetic acid injection.

**P<0.01; significantly different from the control group (N=6).


Central analgesic effect of Karungaliver Kudineer
% increase in reaction time
80
70
60
50
40
30
20
10
0
Control
KVK I
KVK II
KVK III
Standard
Group and Treatment
30min
60min
90min
120min


Peripheral analgesic effect of Karungaliver Kudineer
% Protection from Pain
80
70
60
50
40
30
20
10
0
KVK I
KVK II
KVK III
Standard
Group and Treatment

BIO STATISTICAL ANALYSIS

ANNEXURE IV

BIOSTATISTICAL ANALYSIS

Effect of karungali ver kudineer on HbA1C level (%) in human subjects

S.NO	BEFORE TREATMENT	AFTER TREATMENT
1.	7.6	6.2
2	7	5.8
3	7.8	6.4
4	8.9	7.4
5	8	6.9
6	8.3	6
7	8.2	6.5
8	9	7
9	9.3	7.8
10	9.4	8
11	8	6.5
12	8.6	6.4
13.	8	6.3
14	7.4	6.2
15	8.3	6
16	9	6.8
17	9.6	8.2
18	7.3	6.4
19	8.7	6.8
20	8.5	7.3
21	7.9	6.2
22	8.7	7.5
23	7.1	6.1
24	8.4	6.4
25	7.7	6.9
26	8.9	7
27	7.8	6.4
28	9	7.2
29	9.6	8.1
30	7.4	6.3
31	9.5	7
32	9.6	8.5
33	9.7	9
34	8	6
35	7.2	6.8
36	8.6	6
37	9.3	7.6
38	7.6	6.1
39	8.7	5.9
40	9.8	7.7

Software: spss17 version

Variables: HbA1C levels (%) – before treatment, after treatment

Number of cases: 40

Test: Paired t test

Confidence Interval: 95%

Correlation coefficient (r): 0.774

Before and after treatment mean difference: -1.59±0.54 (%)

P Value (2 tailed): p<0.01

Inference:

The p value is significant (p<0.01). So the treatment was significantly reducing the HbA1C level (%).

Treatment for vatha karsanam

The most popular statistical tool, namely, Fisher's Exact Test analysis has been employed to analyses the effectiveness with the help of a hypothesis.

Hypothesis

There is no reducing symptoms among the patients for the treatment of **vatha karsanam**

Symptoms	**Number of cases**	
	Reduced	**Not Reduced**
BURNING SENSATION NUMBNESS WEAKNESS OF LOWER LIMB	24 88.9%	3 11.1%
GLOVE & STOCKING ANASTHESIA PAIN IN CALF MUSCLE	8 61.5%	5 38.5%

Software: spss17 version

Number of cases: 40

Test: Fisher's Exact test

Confidence Interval: 95%

Result:

P Value (2 tailed): p<0.01

Inference:

Since the p value is significant (<o.o1), The hypothesis is not accepted. So there is significant reduced symptoms among the patients for the treatment of **vatha karsanam** Hence it is concluded that the treatment was effective and significant.

CONSENT FORM

ANNEXURE V

CONSENT FORM

DEPARTMENT OF PG- POTHUMARUTHUVAM

GOVT SIDDHA MEDICAL COLLEGE CHENNAI-106

I certify that I have disclosed all the details about the study in the terms readily understood by the patient.

Date
Signature

Name

Consent By The Patient

I have been informed to my satisfaction by the attending physician for the purpose of the clinical trial and the nature of the drug treatment and follow up including the lab investigation to be performed to monitor and safeguard my body functions.

I am aware of my right to opd out of the trial at any time during the course of the trial without having to give reasons for doing so.

I, exercising my free power of choice, hereby give my consent to be included as a subject in the clinical trial of **KARUNGALI VER KUDINEER** for the treatment of **VATHA KARSHAANA(DM NEUROPATHY)**

Date **Signature**

Name :

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ANNEXURE VI

CASE SHEET
POST GRADUATE DEPARTMENT - BRANCH-I

(POTHU) MARUTHUVAM

CASE SHEET PROFORMA

GOVT. SIDDHA MEDICAL COLLEGE & ANNA HOSPITAL, CHENNAI-106.

CASE SHEET PROFORMA FOR "VATHA KARSANAM"

WARD NO.	:	NATIONALITY	:
O.P. NO/ I.P. NO	:	RELIGION	:
BED NO	:	OCCUPATION	:
NAME	:	INCOME	:
AGE	:	D.O.A	:
SEX	:	D.OD	:

PERMANENT ADDRESS :

DIAGNOSIS :

TEMPORARY ADDRESS:

Govt. Siddha Medical College &

Anna Hospital, Chennai – 106. MEDICAL OFFICER :

COMPLAINTS AND DURATION :

HISTORY OF PRESENT ILLNESS :

HISTORY OF PAST ILLNESS

PERSONAL HISTORY & HABITS:

A. Food : Veg Non veg
B. Marital status :

FAMILY HISTORY

GENERAL EXAMINATION:

1. Physical build : lean normal obese
2. Body weight :
3. Temperature :
4. Pulse rate :
5. Heart rate :
6. Respiratory rate :
7. Blood pressure :
8. Pallor :
9. Cyanosis :
10. Jaundice :
11. Clubbing :
12. Pedal oedema :
13. Lymphadenopathy :
14. Acanthosis nigricans :
15. Hirsutiom :

EXAMINATION OF OTHER SYSTEMS:

- **CARDIO VASCULAR SYSTEM:**
- **RESPIRATORY SYSTEM:**
- **GASTRO INTESTINAL SYSTEM:**
- **CENTRAL NERVOUS SYSTEM:**

1 .MOTOR:

2 .SENSORY:

a. TOUCH -

b .PAIN -

c. TEMPERATURE -

d. POSITION -

e .VIBRATIONS -

f .CORTICAL SENSE -

SIDDHA ASPECTS

Yaakai (udal nilai)	Mukkunam
1. Vatham	1. Sathuva gunam
2. Pitham	2. Raasatha gunam
3. Kapham	3. Thamo gunam
4. Kalappu	

PARUVA KAALAM (SEASONS)	NILAM (PLACES)
1. Kaar Kaalam (Aavani-Puratasi) Aug-sept.	1.Kurinchi (Hills Areas)
2. Koothir Kaalam (Iypasi-Karthigai) Oct-Nov.	2.Mullai (Forest Areas)
3. Munpani Kaalam (Maargazhi-Thai) Dec-Jan.	3.Marudham (Fertile Areas)
4. Elavenil Kaalam (Chithirai-Vaikasi) Apr-May	4.Neithal (Sea Areas)
5. Mudhuvenil Kaalam (Aani-Aadi) Jun-Jul	5.Paalai (Desert Areas)

IYAMPORIGAL/PULANGAL KANMAVIDAYAM

1. Mei (Sensation)
2. Vaai (Taste)
3. Kann (Vision)
4. Mooku(Smell)
5. Sevi (Hearing)

KANMENTHIRIYAM /

1.Kai [Koduthal]
2.Kaal [Nadathal]
3.Vaai [Pesal]
4.Eruvai [Malam Kazhithal]
5.Karuvai [Aananthithal]

MUMMALAM

1. Malam
2. Moothiram
3. Viyaravai

UYIR THATHUKKAL:

Vatham:

1. Pranan
2. Abanan
3. Viyanan
4. Udhanan
5. Samanan
6. Naagan
7. Koorman
8. Kirukaran
9. Devadathan
10. Dhananjeyan

PITHAM:

1. Anal Pitham
2. Ranjaga Pitham
3. Saadhaga Pitham
4. Aalosaga Pitham
1. Prasaga Pitham

KAPHAM:

1. Avalambagam
2. Kledagam
3. Podhagam
4. Tharpagam
5. Santhigam

UDAL THATHUKKAL:

1. Saaram
2. Senneer
3. Oon
4. Kozhuppu
5. Enbu

6. Moolai
7. Sukkilam / Suronitham

Envagai Thervu:

1. Naa -
2. Niram
3. Mozhi -
4. Vizhi -
5. Sparisam
6. Malam
    a. Niram
    b. Nurai
    c. Erugal
    d. Elagal
    e.
7. Moothiram
    a. Neerkuri
        1. Niram
        2. Edai
        3. Manam
        4. Nurai
        5. Enjal
    b. Neikuri
8. Naadi

SIGNS AND SYMPTOMS: PRESENT ABSENT
MORE THAN 5 YEARS OF DM

PAIN IN BOTH EXTREMITIES.

BURNING SENSATIONS IN PALMS &SOLES

TINGLING SENSATION

NUMBNESS

CALF MUSCLE TENDERNESS

FLACCID WEAKNESS ESPECIALLY IN LOWER LIMBS

HIGH STEPPING GAIT

GLOVE & STOCKING TYPE OF ANESTHESIA

Assessment	**BEFORE TREATMENT**	**After Treatment**			
		I	**II**	**III**	**IV**
MORE THAN 5 YEARS OF DM					
PAIN IN BOTH EXTREMITIES.					
BURNING SENSATIONS IN PALMS &SOLES					
TINGLING SENSATION					
NUMBNESS					
CALF MUSCLE TENDERNESS					
FLACCID WEAKNESS ESPECIALLY IN LOWER LIMBS					
HIGH STEPPING GAIT					
GLOVE & STOCKING TYPE OF ANESTHESIA					

LABORTORY INVESTIGATIONS:

		BT	**AT**
1.Blood	Tc		
	Dc		
	ESR		
	Hb		
	Bl-sugar (F)&(PP)		

Bl.Urea

Sr.Cholesterol

Sr.Creatinine

2.Urine - alb

Sug

Dep

3 .MONO FILAMENT TEST:

4 .NERVE CONDUCTION STUDY:

TRAIL DRUG:

KARUNGALI VER KUDINEER

Dose:

30ml. twice a day before food…

Duration of treatment

Pathiam (Do's and Don'ts

Prognosis at the end of the treatment

Medical Officer Signature: **H.O.D**

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BIBILIOGRAPHY

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- SIDDHA MARUTHUVAM
- SIDDHA MARUTHUVAANGA SURUKKAM
- NOI NADAL NOI MUDHAL NADAL
- ANUBAVA VAITHIYA DEIVA RAGASIYAM
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